University of Alberta

The health impact of climate change at Tell Leilan (Syria): a multi level analysis of developmental enamel defects

by

Leslie Dawson

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A thesis submitted to the Faculty of Graduate Studies and Research
in partial fulfillment of the degree of Master of Arts

Department of Anthropology

Edmonton, Alberta

Fall, 1999

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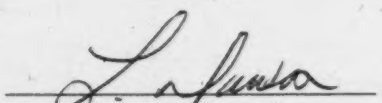

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "The Health Impact of Climate Change at Tell Leilan (Syria): A Multi-Level Analysis of Developmental Enamel Defects" submitted by Leslie Dawson in partial fulfillment of the requirements for the degree of Master of Arts.

Dr. Nancy Lovell

Dr. Owen Beattie

Dr. Jeremy Rossiter

Dedication

I would like to dedicate this thesis to the memory of my father,

Ronald Sanford Dawson (1933-1998),

who taught me the importance of hard work, honesty and respect.

Abstract

To test the hypothesis that climate change caused the abandonment of Tell Leilan during the late 3rd millennium BC (Weiss et al. 1993), 153 permanent teeth were assessed for developmental enamel defects as indicators of non-specific stress. Although the results indicate a greater frequency of hypoplasia for the pre-abandonment period, whether there is any difference in the distribution of stress within the population is unclear. The higher stress involves a greater number of physiological insults experienced by individuals, and may include stress of a greater duration and/or severity. Data from the analysis of hypocalcification contradict those for hypoplasia, and may reflect pseudo-opacities derived postmortem, or antemortem conditions producing *acquired* rather than *developmental* defects.

Acknowledgements

I would like to thank my supervisor, Dr. Nancy Lovell, for her support and direction, the members of my committee, Dr. Owen Beattie and Dr. Jeremy Rossiter, and Dr. Pam Willoughby for chairing my defense. I am also grateful to Dr. Harvey Weiss of Yale University for making the Tell Leilan sample available for study, and Scott Haddow for the photograph of enamel hypoplasia.

A special thanks goes out to my family and friends, especially Dan, for their support and availability for a pint.

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Chapter 1:
Introduction

Archaeological investigations of the multi-period site of Tell Leilan have produced information pertaining mostly to the rise of indigenous urbanization for the site, as well as the Habur Plains, and to its subsequent collapse (Weiss 1983; 1986b; 1990a; 1990b; 1992; 1996; Weiss et al. 1993; Wright 1998). Weiss et al. (1993) propose climatic change, coinciding with a volcanic eruption, that caused an increase in aridity substantial enough to affect agricultural production as the catalyst for the abandonment of the site. This thesis will employ another form of evidence to explore this claim; the analysis of dental enamel defects as indicators of non-specific systemic stress.

Anthropologists interpret the presence of developmental defects of enamel as representing systemic stress during the period of tooth crown formation (i.e. childhood). Systemic stress is generally defined as inadequate diet (e.g. malnutrition) or infectious disease, or the synergistic relationship between the two: a disease condition may disrupt an individual's normal metabolism, while malnutrition may leave an individual more susceptible to disease. The effect of such stress is a disruption in dental enamel formation at the cellular (ameloblastic) or microstructural (crystallographic) level and will result in macroscopic defects known as enamel hypoplasia and/or hypocalcification. These defects are, therefore, developmental and have been proven through experimental studies to be sensitive, but non-specific, markers of systemic stress (Kreshover 1960). Such non-specificity translates into an inability to determine a direct etiological cause but designates such defects as markers of stress on an individual and allows for the construction of a stress profile for the population.

The analysis of developmental enamel defects has been commonly used to detect changes in health patterns and stress profiles in archaeological populations (for reviews of past studies see Goodman and Rose 1991, and Skinner and Goodman 1992). Issues pertaining

to culture change can be addressed as to their influence on human health. For example, numerous researchers found an increase in the frequency of enamel defects with the transition from hunter-gatherer to agriculture (see Cohen and Armelagos 1984), while others have investigated the effect on health of contact between the Europeans and the indigenous populations of the New World (Hutchinson and Larsen 1990; Ubelaker 1994).

This thesis will address the claim of Weiss et al. (1993) that climate change was fundamental to the collapse of the society at Tell Leilan. Through the analysis of developmental enamel defects as indicators of non-specific stress, the effect of climate change on health will be investigated. If climate change, as opposed to political or social factors, did indeed provide the impetus for the collapse of the civilization, then this should be evident in not only an increase in the frequency of individuals exhibiting defective enamel but in the distribution of stress indicators within the population as well.

To begin with, the necessary background information for this study will be provided in Chapters 2 and 3. Chapter 2 will review the dynamic culture history of the site of Tell Leilan, and its relationship to the pattern of health and stress over the various cultural periods. Chapter 3 will discuss current knowledge of developmental enamel defects as indicators of non-specific stress, as well as potential methodological problems presently faced by anthropologists. The frequency and distribution of hypoplastic and hypocalcified defects will be compared in Chapter 4, in order to address issues concerning the use of enamel hypocalcification as an indicator of non-specific stress. Chapter 5 will address the health impact of abrupt climate change at Tell Leilan through a multi-level analysis of enamel hypoplasia. This analysis will also include a methodology employed to specifically address a comparison of hypoplastic defect frequency. Finally, Chapter 6 will summarize what has been learned in this research and conclude with a brief discussion on the effects of climate change on health at Tell Leilan.

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Chapter 2:
The dynamic culture history of Tell Leilan (Syria) and its potential effects on health and stress

Introduction

Scholars increasingly believe that the archaeological evidence of widespread site abandonment at the end of the third millennium BC signals the collapse of civilizations from southwest Europe to central Asia (Weiss 1996; Weiss et al. 1993; Wright 1998); the downfall of the Early Bronze Age urban culture in Palestine, the end of the sixth and last dynasty of the Old Kingdom in Egypt, the decline of the Indus Valley civilization in India, the disruption of the settlements on Crete and the Greek mainland, and in northern Mesopotamia, the end of the Akkadian occupation (Ben-Tor 1992; Weiss 1996; Weiss et al. 1993; Wright 1998). The underlying cause of this far-reaching phenomenon has been a subject of debate among historians and archaeologists. Often, researchers cite political or social sources, such as destruction by invading nomads or barbarians, or attrition through constant warfare, as the cause of such societal breakdown (e.g. Ben-Tor 1992). An alternative explanation is ecological: a change in climatic conditions directly affecting agricultural subsistence and/or living conditions (e.g. Rosen 1989, 1995; Weiss et al. 1993).

For northern Mesopotamia, in particular, an inability of the Akkadians to adapt to the social network of the northern cities, or an invasion by Amorite nomads (Weiss 1996), have been cited as instrumental in the collapse of northern society. In reference to the widespread nature of the collapse, Weiss et al. (1993) propose an ecological model to explain the abandonment of the northern Mesopotamian site of Tell Leilan: climate change, commencing at ~2200 BC, and lasting for a period of approximately 300 years, denied viable agriculture and was, consequently, the impetus for the collapse of the society.

By reviewing the history of the site, aspects of culture change can be discussed as to their possible influence on the health of the inhabitants of Tell Leilan. Within this framework, the health effects of climate change can be assessed relative to past cultural events to determine the potential impact. Abrupt or severe changes in climatic conditions could affect subsistence or social stability or both. If Weiss' hypothesis is correct, then the effect of such environmental change should be revealed in a higher frequency of enamel defects for the period of occupation prior to abandonment of the site than seen in previous periods.

The culture history of Tell Leilan

Mesopotamia (Greek: the land between the rivers) is the ancient area defined by the Euphrates and Tigris Rivers and their tributaries in western Asia (Bahn 1992). Made up of two general geographical regions, the northern piedmont and the southern alluvial zone, Mesopotamia maintained different regional climates which subsequently influenced cultural development and adaptability. The dividing line between north and south Mesopotamia runs approximately between modern Hit and Samarra and delineates the uppermost limit of the alluvial zone (Lloyd 1984). North of this line, the Euphrates is more restricted to the valleys of the Jezirah plateau (Figure 2.1).

The earliest known cultures of Mesopotamia relied on basic farming economies at the village level. However, as the communities and cultures developed, each region adapted to their environment: the rain-fed north practiced dry-farming whereas different ecological conditions in the south led to the development of irrigation-farming. Evidence of irrigation in the south has traditionally led archaeologists and historians to consider southern Mesopotamia as the "cradle of civilization" with Sumerian and Akkadian cultures as the main focus. Any development of complex or urban society in the form of cities and states in northern Mesopotamia had to have come via cultural diffusion, migration or imperialism out of the south. Recently, however, new data recovered from

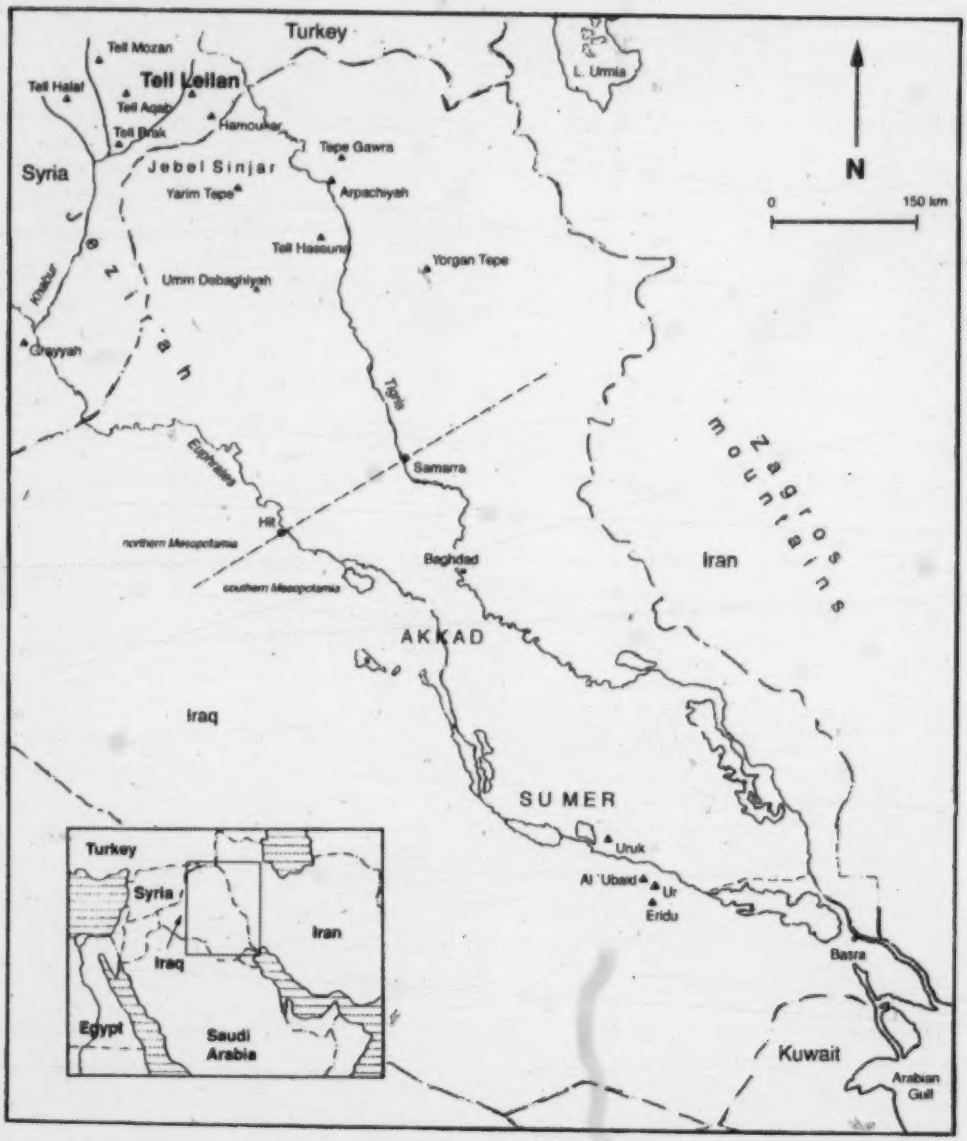

Figure 2.1: Map showing north and south Mesopotamia and identifying sites, rivers and regions mentioned in the text (● = modern cities, ▲ = archaeological sites) (inset adapted from Bernbeck 1995:10; map adapted from Lloyd 1984:14).

numerous sites in the north have challenged this traditional assumption and provided evidence of an indigenous development of complex society and urban settlement in

northern Mesopotamia (e.g. Weiss 1986). One such site, through occupation over several millennia, provides insight into the development and influences upon northern Mesopotamian settlements leading from village-based political and economic structures to the emergence of cities and states, as well as the possible factors involved in settlement decline in northern Mesopotamia during the late 3rd millennium BC.

The multi-period site of Tell Leilan

Leilan is a multi-occupational tell site situated on the rain-fed Habur plain of northeastern Syria. Located on the left bank of the perennial Wadi Jarrah, Tell Leilan rests in the dry-farming region of what is referred to as the Habur Triangle (Weiss 1991). The massive walls of the tell rise 15 meters above the plain and the site encompasses an area of more than 90 hectares, making Leilan "...one of the largest ancient sites in northern Mesopotamia..." (Weiss 1985:6).

Under the direction of Harvey Weiss, and in cooperation with the Directorate-General of Damascus, Yale University began its investigation of Tell Leilan in 1978 with an initial topographic survey of the site (Weiss 1985; 1991b). Subsequent archaeological field seasons, focusing for the most part on the Acropolis and the Lower Town, have produced a detailed understanding of the 3rd to 2nd millennium BC occupational history of the site, as well as a new understanding of the rise of complex societies (e.g. Schwartz 1987) and cities (e.g. Weiss 1983; 1990a; 1990b) on the Habur plain.

Archaeological investigation of Tell Leilan has included vertical soundings, horizontal exposures and regional surveys (Figure 2.2). The periods of increased social complexity and urbanization, beginning with the late Ninevite V and state formation through the Akkadian occupation and subsequent abandonment of the site, have been thoroughly investigated archaeologically through horizontal exposures in the Lower Town and Acropolis. The earliest cultural occupations, the Ubaid (Leilan VI) and Uruk periods (Leilan IV and V), have had limited exposure through soundings at the Acropolis. Due to

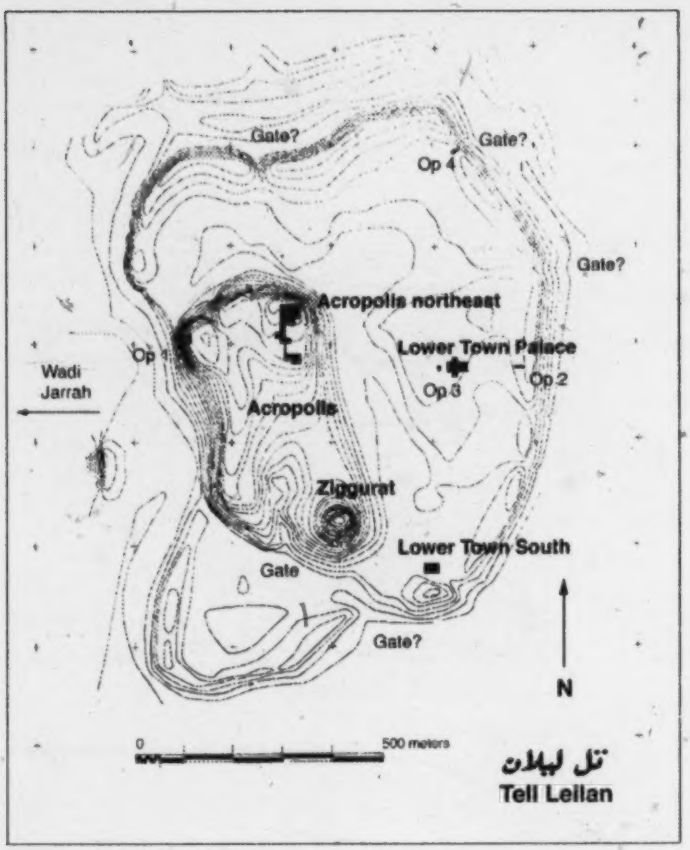

Figure 2.2: A site map of Tell Leilan identifying the Acropolis, the Lower Town and various archaeological features as well as the different excavation operations (map adapted from Weiss 1991:704).

their limited exposure, the earlier periods will be supplemented with cultural information from contemporary northern Mesopotamian sites whenever possible.

In terms of cultural horizons, most periods are defined by means of ceramic traditions: the emergence, presence, frequency and disappearance of various pottery styles and shapes delineate one period from the other. Although these "ceramic horizons" are most likely a survival of the use of pottery in the relative dating method of seriation, they have been

subsequently dated absolutely (see Weiss et al. 1993). The culture history of Tell Leilan is represented by general periods, Ubaid, Uruk, Ninevite V, the Tell Leilan urban period, Akkadian imperialism and the reign of Shamshi Adad. Each general period is further divided into subphases relating to culture change or development following the Leilan chronology (e.g. Leilan period IIId as secondary state formation) (Table 2.1). To begin with, the earliest periods noted in northern Mesopotamia, the Hassuna and Halaf cultures, will be briefly reviewed to provide a cultural basis for the Ubaid (i.e. the earliest cultural horizon recognized at Tell Leilan).

Hassuna and Halaf cultures

The Hassuna culture (~ 6th millennium BC) is often referred to as the "village period" in northern Mesopotamia (Bernbeck 1995) and generally coincides with the late Neolithic (Lloyd 1984). Named after its first recognition at the site of Hassuna on the Tigris river, it has been identified at sites across northern Mesopotamia (Finegan 1979). This period reveals simple rain-fed agricultural practices with the cultivation of wheat and barley, and a mix of animal husbandry (sheep, goat and cattle) and hunting (Schwantes 1965; Lloyd 1984). Architectural remains reveal complexes of small rooms surrounding open spaces. Changes in the ceramic tradition demarcates the Archaic from the Standard Hassuna and reveals pin-scratched or painted, unburnished pottery in a limited range of bowl and jar shapes (Lloyd 1984).

The succeeding Halaf (~ late 6th to early 5th millennium BC), represented geographically by the presence of painted pottery with geometric, floral and naturalistic motifs, is found across northern Mesopotamia and Anatolia at sites such as Arpachiyah, Tell Aqab, Yarim Tepe 2 and Tepe Gawra. Its original identification was at the site of Tell Halaf on the Habur river in northeastern Syria (Finegan 1979; Schwantes 1965). The Halaf culture extended from the Euphrates and Habur rivers to modern Mosul in Iraq (Finegan 1979) with no real equivalent cultural complex found in southern Mesopotamia at this time (Lloyd 1984). Settlements are uniformly small (<4 hectares) and characterized by small,

Table 2.1: Chronology of Tell Leilan including general periods, Leilan periods, radiocarbon dates and subphases (based on Bahn 1992; Weiss 1990a; Weiss et al. 1993)

General period	Leilan period	Date (BC)	Subphase
Resettlement	I	~1990-1728	reign of Shamshi-Adad
Habur hiatus 1	-----	~2200-1900	desertification and desertion
Akkadian imperialism	IIb	~2300-2200	occupation
Urban period	IIa	~2400-2300	consolidation of complex state administration
Ninevite V			
late	IIId	~2600-2400	secondary state formation
middle	IIIb-c	~2900-2600	pre-state occupation
early	IIIa	~3000-2900	Uruk collapse
Uruk			
late	IV	~3300-3000	late Uruk expansion
early	V	~4100-3300	early Uruk
Ubaid	VI	~5500-4100	northern Ubaid
Halaf	-----	~late 6th-early 5th millennium	early and late Halaf
Hassuna	-----	~6th millennium	archaic and standard Hassuna

circular buildings (referred to as *tholoi* after the Greek term for similar tomb architecture) made of *pisé* or mud plaster and are considered to be domestic dwellings (Bernbeck 1995; Lloyd 1984). Subsistence involved dry-farming of wheat and barley, and herding of sheep, goat, cattle and pig. It is believed that this period coincides with the emergence of chiefdoms (Fagan 1989).

Ubaid period

The earlier phases of the Ubaid are restricted to southern Mesopotamia whereas the later phases are found in northern Mesopotamia and throughout western Asia through a system of long-distance trade and local exchange. The Ubaid period is considered the first

example of the dry-farming north and the irrigation-based south sharing the same culture complex (Sürenhagen 1986). Southern Ubaid culture reveals evidence of irrigation to extend cultivable land (Schwantes 1965) and the presence of ceremonial architecture such as the temple at Eridu (Fagan 1989). Archaeological remains in northern Mesopotamia exhibit a similar river-oriented settlement structure with a trend towards increased sedentism and population densities. Architecture included temple architecture, as evident at Tepe Gawra, and metallurgy was not specialized, as seen in the handful of hammered or cast pure copper items recovered from Tepe Gawra (Lloyd 1984).

Archaeological evidence from Tell Leilan has identified a northern Ubaid occupation in the form of domestic architecture through a vertical exposure (i.e. a sounding on the Acropolis) (Weiss 1983). The Northern Ubaid is represented by the Leilan VI period (~5500-4100 BC) and is thought to coincide with the broader Chalcolithic time period (Lloyd 1984)

Uruk period

The Uruk period is traditionally associated with the development of urban societies in southern Mesopotamia and is characterized architecturally by the *ziggurat*, the stepped temple pyramid (Fagan 1989) and the "middle hall house" (Sürenhagen 1986:9). As Schwantes (1965:22) explains, the period sees a shift from village to "temple-city" with the development of a writing system to record economic transactions, and the use of cylinder seals for temple administration. It is during the late Uruk period, or "Uruk expansion", that this culture appears outside of southern Mesopotamia (Bahn 1992).

Evidence of the Uruk period in the northern plains indicates centralization and specialization (Weiss 1983). Distribution of settlements is similar to the preceding Ubaid period with Uruk sites found along the Tigris, Habur and Balikh Rivers. The estimated size of several Uruk sites (e.g. Tell Brak ~40 hectares and Hamoukar ~90 hectares) is considered to suggest urban control of surrounding dry farming areas by these larger centers (Sürenhagen 1986). Architecture reveals temples, such as the Eye temple found at

Tell Brak, and the first traces of wheel-made pottery seen in the monochromatic burnished ceramic tradition characterized by bevel-rimmed bowls. Burial artifacts reveal a greater interest in metallurgy with evidence of alloys (e.g. bronze) (Lloyd 1984). Excavations from Tepe Gawra reveal that mortuary practices become more sophisticated during the Uruk period: although infants remain buried beneath floors, various distinguished members of the community are interred in carefully built stone or mud-brick tombs, in a contracted position, with numerous burial artifacts including gold and items reflecting long distance trade (Lloyd 1984). It is during the late Uruk expansion that exogenous urbanization of the Habur Plains begins (Weiss 1990a) with different settlement types emerging: sites are either classified as "genuine Uruk settlements" (e.g. Tell Brak and Grayyah) or as "Uruk-related" (e.g. Tell Leilan, Tepe Gawra and Yorgan Tepe) (Sürenhagen 1986:10,13).

Two cultural horizons represent the Uruk period at Tell Leilan: Leilan V (~4100-3300 BC) indicates the early Uruk and Leilan IV (~3300-3000) equals the late Uruk expansion. Material culture recovered from a sounding at the Acropolis suggests a general continuance of ceramic ware and shape in the early Uruk, as seen in the late northern Ubaid, but with a decrease in the frequency of painted ware (Weiss 1983).

Ninevite V period

The transition from the Leilan IV to Leilan IIIa is distinguished by the disappearance of characteristic Uruk pottery and its replacement by painted ware of the Ninevite V culture (Weiss 1983; 1990b). The term Ninevite V represents both a ceramic tradition as well as an archaeological horizon in northern Mesopotamia (Bahn 1992).

The collapse of the Uruk urban culture is represented by the Leilan period IIIa (~3000-2900 BC). The region sees a return to a dispersed settlement pattern of small, low density communities, as seen in the limited distribution of Ninevite V settlements around Tell Leilan (the site itself is approximately 15 hectares) (Weiss 1983; 1990a). Two burials recovered from the Leilan IIIa period housed ceramic vessels but are without burial

artifacts indicative of long distance trade or craft specialization (Schwartz 1986), and may be indicative of a lesser stratified society.

Leilan period IIIb-c (~2900-2600 BC) is considered the pre-state Ninevite V occupation and provides possible insight into increasing social complexity. Excavations at Tell Leilan have revealed a high status burial covered by a brick structure dating to the Leilan IIIc period which may indicate rank in the society: artifacts found in association with the burial include a necklace of carnelian beads and shells, two copper pins, a cylinder seal and a cache of thirty-two ceramic vessels in an associated pit as well as four vessels around the burial itself (Schwartz 1986).

Leilan IIId (~2600-2400 BC) correlates to the late Ninevite V ceramic tradition and is considered a time of secondary state formation with regional and settlement changes indicating increased centralization and specialization (Weiss et al. 1993). During this period, there is evidence of a region-wide change in settlement organization and site size across the Habur Plains. The small, dispersed agricultural communities gave rise to indigenous state-level societies in which central authorities organized a geographical network to ensure efficient collection, storage and redistribution of resources (Weiss et al. 1993). Three major urban centers (Tell Leilan, Tell Brak and Tell Mozan), each encompassing ~100 hectares, developed equidistant from each other on the perennial Jarrah, Jaghjagh and Khanzir drainages. Weiss (1992) proposes two theories about the equidistance of these centers that may provide insight into the structure of third millennium BC northern Mesopotamian agricultural state systems. One theory discusses the equidistance of the sites as reflecting regional control of up to 25 kilometers of rain-fed territory around each center, defined by regional populations and hinterland productivity. Each center would have been supported by a three or four-tier system of secondary centers which would optimize agroproduction and transportation (Weiss et al. 1993). Another theory considers that the regional layout may also reflect a form of agricultural organization favoring stream location, rainfall, soils and topography in response to changing climatic conditions seen in a shift from regular alluviation to

increased seasonality of water discharge evident in paleoclimatological analyses (Weiss et al. 1993).

Stein (1994:14) discusses the increase in settlement size of Tell Leilan, from the pre-urban period ~15 hectare site to the urban period ~90 hectare city, as related to amount of land needed for cultivation to support the population (i.e. "estimated minimum sustaining areas") (Figure 2.3). The author concludes that the circumscription of Tell Leilan suggests that "...the urban center lacked sufficient land to meet its subsistence needs..." and would, therefore, need secondary centers to supply agricultural surplus to Leilan.

The increase in site size saw the transition of a ~15 hectare Acropolis-based community into a ~90 hectare urban settlement encompassing the Lower Town. Excavations of the Lower Town noted construction on virgin soil, attesting to the expansion of Tell Leilan during this period, and provided evidence of careful, central-based planning. The Lower Town settlement included a street, 4.75 meters wide which was lined on each side by mud-brick walls. On either side of the walls were domestic structures that opened onto alley-ways. Walls running perpendicular to the street are considered by Weiss et al. (1993) to delineate residence or property lines. This area was continuously rebuilt and occupied until the abandonment of the site at the end of the Leilan period IIb (Weiss 1990b). Further evidence is noted with the excavations at the northeast aspect of the Acropolis where a palace with storerooms and public doorways into adjacent chambers has been uncovered (Weiss 1992).

The transition from pre-state Leilan IIIc to Leilan IIId also sees the transformation of a simple village economy to a system of centralized collection, storage and redistribution. Architectural remains reveal the replacement of small scale domestic structures on the Acropolis with a block of rectangular storerooms of approximately 200m^2 and the

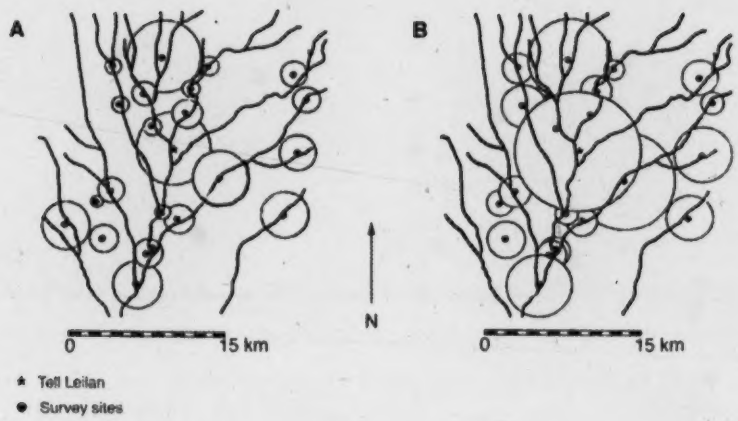

Figure 2.3: Diagram showing the amount of land (defined by the circles) needed for cultivation to support settlement size (A = preurban Tell Leilan; B = urban Tell Leilan). The circumscription of Tell Leilan is considered to suggest the need for secondary centres to supply agricultural surplus to Leilan (adapted from Stein 1994:14).

recovery of numerous clay jar sealings points to a central administration (Weiss et al. 1993). The pre-state to state transition is also considered indicative of a society undergoing a transformation from a ranked to a class based society (Weiss 1991), however mortuary data reveals too small a sample size to statistically attest to or disprove the development of urban stratification (Schwartz 1986).

Material culture remains suggest changes in production and specialization. Cereals consumed include barley (*Hordeum vulgare*), emmer wheat (*Triticum dicoccum*) and durum wheat (*Triticum durum*). Analysis of the botanical remains suggests local processing of large quantities of cereals for storage, evident in the presence and frequency of inedible byproducts (Weiss et al. 1993). Faunal remains see an almost equal exploitation of pig (33%), sheep and goat (32%) and cattle (29%) (Weiss et al. 1993). Metallurgical analysis of bronze pins recovered from a burial context provide the of earliest evidence at Tell Leilan of tin bronze as opposed to arsenical copper and indicate further specialization (Weiss 1991b).

The Tell Leilan Urban Period

The transition from the Leilan IIId period to the Leilan IIa (~2400-2300 BC) sees the alteration of economic and social organization and a time of "...consolidation of complex state administration of the cities of Subir..." (Weiss 1990a; Weiss et al. 1993:999). The construction of a 2 meter wide defensive wall around the Acropolis housing the storerooms and administrative buildings at Tell Leilan and the appearance of numerical notations (based on circles and vertical lines) on the rims of large cereal storage vessels reveals state organizational change (Postgate 1994; Weiss et al. 1993). Further organizational change is seen in the first appearance of mass-produced pottery and an abandonment of the labor-intensive craft of incising on ceramics. Faunal remains provide evidence of horse, mule and large onager for the wheel-drawn transport of the cereal harvest (Weiss et al. 1993).

Akkadian imperialism

Further political change occurred at Tell Leilan during the Leilan period IIb with Akkadian imperialism. At its greatest the Akkadian empire spanned 800 miles from the Persian Gulf to the headwaters of the Euphrates in Turkey (Gibbons 1993). From ~2300-2200 BC, southern Mesopotamia was united under the rule of Sargon of Akkad and his descendants. The Akkadians first ruled the irrigation-based agriculture of southern Mesopotamia and subsequently expanded into northern Mesopotamia and intensified rain-fed agriculture. Documentation from Tell Brak indicates political control of Tell Leilan and Tell Mozan by the Akkadians via Tell Brak (Weiss et al. 1993).

Senoir and Weiss (1992; Weiss 1991b) discuss four features which reflect the effects of Akkadian imperialism on Tell Leilan; regional population centralization, city wall construction, ration-fed labor, and channelization. A redistribution and centralization of population is evident with people concentrating within centers such as Tell Leilan. Part of this redistribution sees an apparent removal of second-tier centers and the elite from production administration and the maintenance of villages for imperial agricultural

production (Weiss et al. 1993). Although people concentrated within Tell Leilan, archaeological evidence suggests settlement size to have changed from ~90 hectares to ~75 hectares for the period IIb city (Weiss 1990a). Part of the decrease in settlement size may have involved people moving within the newly constructed city wall for protection thereby increasing settlement density.

Fortification of the site under Akkadian occupation sees the first evidence for a city wall at Tell Leilan. Part of this fortification included an earthen rampart on the northern side of the city where "...a natural depression and rise afforded protection..." (Weiss et al. 1993). The defensive nature of the wall is seen in its massive size. The wall was made up of three parts; the inner and outer walls were 8 meters wide and the middle wall was a meter (Weiss et al. 1993). Construction of this city wall reflects not only the need for defense, but central planning and the ability to mobilize workers as well.

Worker food rationing is evident in two forms of data. Paleobotanical remains from floors of houses and courtyards reveals pre-cleaned cereals with little inedible portions. The absence of cereal processing by-products reflects the distribution of processed cereal foods and is considered by Weiss et al. (1993) to suggest imperial labor ration payments to the households of the Lower Town south (Weiss et al. 1993). Further evidence of labor rations is seen in the recovery of standard-size ceramic vessels (0.33-, 1- and 1.5-litres), referred to as "sila bowls", which only occur during this period (Blackman et al. 1993; Senior and Weiss 1992). Weiss et al. (1993) propose that these standardized vessels held worker rations of barley and oil based on epigraphic documentation.

In response to a continuing decrease in water discharge and siltation initiated in the late Ninevite V period, there is evidence at Tell Leilan for the stabilization of water ways through channelization and repeated clearing (see Weiss et al. 1993). Weiss et al. (1993) interpret this strategy as reorganization of production and as reflecting Akkadian expertise in water management developed in southern Mesopotamia with irrigation farming.

The archaeological evidence and late Akkadian period documentation points to a prosperous culture with a successful imperial economy which sustained long-distance trade, the construction of monumental buildings and massive agricultural projects, and a substantial military (Weiss et al. 1993). The Akkadians held their politico-military control over the Habur plains until ~2200 BC when the site of Tell Leilan and numerous other northern Mesopotamian sites were simultaneously deserted.

Desertification and desertion

At ~2200 BC, Akkadian imperialism at Tell Leilan collapsed and the site was abandoned. This marked the beginning of an "occupational hiatus" at Tell Leilan that lasted until reoccupation during Leilan period I (~1900 to 1728 BC). This hiatus is evident in the archaeological soundings throughout the site and surface surveys have found no evidence for ceramic assemblages dating to this period (Weiss et al. 1993:999). Weiss (1992) notes that a similar abandonment is evident at almost all contemporary sites across the Habur and Assyrian plains and that there is evidence of disturbed settlement and political systems from southeast Europe to southern Mesopotamia. The abandonment period is referred to as the "Habur Hiatus 1" and this hiatus, Weiss et al. (1993:999) argue, coincides with a period of climatic change.

Evidence of climatic change at Tell Leilan

The combination of an episode of tephra fall and a subsequent phase of marked aridity, "...which seems to match, in amplitude and duration, a major short-term climate change...", is considered fundamental by Weiss for the abandonment of Tell Leilan (Weiss et al. 1993:1001). The tephra fall, referred to as Abu Hgeira 2 tephra, is indicative of a volcanic eruption, although identification of the volcano itself and chronological proof of its eruption are not yet clear. A volcano within the Anatolian-Caucasian area is considered, however, to be the source of the Habur Plains ash but the chronology of volcanic activity during the Holocene for this region is not well understood (Weiss et al. 1993).

Evidence of climate change has been confirmed, however, through soil and sediment analyses. Weiss et al. (1993) discuss paleoenvironmental data found within a well defined stratum in the Lower Town South and divide the depositional information into three phases (for a more detailed review of the soil and sediment analysis see Weiss et al. 1993);

> Phase 1: The early abandonment phase is represented by an aeolian deposit containing a mixture of fine silt-sized volcanic glass fragments and calcitic clay loam derived from surrounding collapsed mudbrick structures. Analysis reveals soil moisture was high enough to maintain bioturbation (e.g. earthworm activity) and to produce slight changes in the volcanic ash. Rainfall was sufficient to disintegrate existing mudbrick structures.
>
> Phase 2: The intermediate phase consists of a 20 centimeter thick deposit of "...grey well -rounded, sand-sized pellets and loose silt-sized calcitic silt, and abundant very fine fragments of weakly weathered volcanic glass..." (Weiss et al. 1993:1000). Analysis of this deposit indicates local strong wind deflation and long-distance aeolian transport, a decrease in soil bioturbation, a simultaneous reduction in rainfall and occasional erosion of surface soil by violent rainstorms. The soil and sediment analyses also point to marked aridity induced by intensified wind circulation and an apparent increase in dust veil frequency.
>
> Phase 3: Analysis of the final phase notes an increase in soil moisture and bioturbation. Furthermore, this phase sees indications of soil augmentation, a re-establishment dry-wet seasonal contrast, a decrease in rainstorm activity, and progressive stabilization of the soil.

Although analysis of these deposits does suggest climatic change in the form of marked aridity, the link between the tephra fall and the increase in aridity is not clear (Gibbons 1993). In a discussion of "climatic effectiveness" of volcanic eruptions, Pyle (1992:125) notes that although a connection between volcanic eruptions and proxy phenomenon (e.g.

evidence of climate change in ice-cores and tree-rings) has been demonstrated, "...volcanoes are merely one of several agents which modulate surface temperatures...". Otterman and Starr (1995:128) address the Mesopotamian drought of 2200-1900 BC through a discussion of surface and climatic conditions. The authors propose that the volcanic activity deposited sufficient dust to trigger a transition from one surface condition to another which may have subsequently altered climatic conditions. The tephra fall would have smothered existing vegetation (i.e. agriculture) "...opening the region to saltation from adjacent hyperarid zones..." resulting in unstable soil conditions. These new soil conditions may have lead to a decrease in mean annual rainfall denying agricultural activity until an event or number of events caused surface conditions to revert back to pre-2200 BC soils and vegetation. However, Weiss et al. (1993; Weiss 1996) note that the climatic change may not be a direct response to volcanic activity (for a discussion see Weiss et al. 1993) and instead stress that the causal link between contemporary site abandonment and climate change is evident in neighboring regions.

Archaeological and historical data pertaining to the abandonment of Tell Leilan

Archaeological investigations have revealed a trend of political instability throughout the greater region. Weiss et al. (1993:1002) point out that the climate change and Akkadian collapse are "...synchronous with climate change and collapse phenomena documented in the Aegean, Egypt, Palestine and the Indus..." (e.g. Esse 1989; Rosen 1989, 1995). For example, in reference to the decline of urban cultures of Palestine, Ben-Tor (1992:123) considers the Early Bronze Age IV as "...the phase during which the process of the desertion of towns reached its peak...". This period (~ 2350-2000 BCE), contemporary with the Habur Hiatus 1, not only sees an abandonment of large tell sites, but a shift of population to more marginal rural areas in the Jordan Valley and the Negev desert, and a transition from intensive agriculture to pastoralism and small-scale mixed agro-pastoralism (Dever 1995). In a discussion of the reorganization and abandonment of Early Bronze Age sites, Rosen (1995) provides four lines of paleoclimatological evidence indicating a shift to a drier climate coinciding with this period (~ 2200 BC). Rosen (1995), however,

does not support the view that climate change is solely responsible and discusses the social response to environmental change as part of a multicausal perspective of societal collapse.

The ultimate social response to changing conditions in northern Mesopotamia has been documented historically with evidence of a massive migration into southern Mesopotamia occurring at the 300 year period of abandonment. Indeed, Weiss (1986; Weiss et al. 1993) suggests the displacement of ~14,000 to ~28,000 people from within a 15 kilometer region of Tell Leilan, including Hurrian, Gutian and Amorite populations. This may be reflected in the translations of clay tablets that reveal an increase in northern tribal names in southern cities (Gibbons 1993). Gibbons (1993:985) notes that a migration of people from north to south would have put a strain on the food and water supplies "...to the point of civic collapse...". Furthermore, scribes from the Third Dynasty of Ur describe an "...influx of northern 'barbarians'..." (Gibbons 1993:985). Weiss (1996) proposes that with the collapse of northern cities, the Akkadian Empire would have lost much of its wealth directly affecting the southern Mesopotamian economy and leaving Akkad vulnerable to invading barbarians.

Period I: resettlement and the reign of Shamshi-Adad (~1900-1725)

Reoccupation of Tell Leilan occurs during ~1990-1725 BC (Leilan period I) and coincides with a return to pre-tephra fall climatic conditions. Excavations of second millennium temples on the Acropolis and the Lower Town palace provide evidence of the largest site on the Habur Plains under centralized Amorite control (Weiss et al. 1993).

In contrast to the Leilan period II city, archaeological evidence does not suggest a densely inhabited city, but rather "hollow" cities and a dense distribution of village settlements (Akkermans and Weiss 1989; Weiss 1986). Through the analysis of recovered administrative artifacts, such as cylinder seal impressions (Parayre 1989) and cuneiform Tablets (Eidem 1989), the identification of the site as historic Shubat En-lil and the surrounding region as Apum has been essentially confirmed (Akkermans and Weiss 1989; Weiss 1991). The palace is considered to have been constructed during the reign of

Shamshi Adad I of Assyria and rebuilt and renovated through successive rulers (Akkermans and Weiss 1989).

Discussion

In reviewing the culture history of Tell Leilan, several themes of change are evident; namely, changes in settlement size and density, social complexity, and climatic conditions. Since each of these changes could produce stress on the individual and/or population in terms of dietary inadequacy, infectious disease or both, all three could produce defective dental enamel.

Changes in settlement size and density would effect the potential pathogen load of the environment as well as the likelihood of infectious disease transmission and, would therefore, alter the *frequency of stress*. A shift to a more aggregated or dense settlement would allow for infectious pathogens to exist in crowded living conditions, as well as facilitate disease transmission, whereas a dispersed settlement or population would lessen the pathogen load. For instance, in a biomedical study of the effect of a shift from a relatively dispersed to an aggregated sedentary settlement pattern on the health of Kalahari hunter-gatherers, Kent and Dunn (1996) note an increase in infectious disease despite adequate diets. At Tell Leilan, an increase in site size and settlement density occurs during the late Ninevite V with settlement reorganization and, similarly, during Akkadian imperialism with the population moving inside the city wall decreasing overall site size but in turn increasing population density. Both changes in settlement could increase the potential pathogen load. Conversely, a shift to a more sparse distribution of smaller settlements, such as the collapse of the Uruk urban culture, would lessen disease transmission.

Alterations in social complexity, such as increased stratification or hierarchy, would shift the *pattern of stress* within the population. In a discussion of the social response to environmental change, Rosen (1995) explains that in complex societies, different segments

of the society are unequally affected by drought or famine. Citing Rao (1974) and Spitz (1980), Rosen (1995:37) notes that the elite may be better off during a famine year since they may have "...larger reserves of non-perishable items that can be traded for food, as well as freer access to public reserves of food...". For example, with the emergence of classes during the urban Leilan period IIa or with occupation under the Akkadians, different segments of the society would have different access to resources and, therefore, different potential stresses. For example, the peasant class may have had unequal access to food resources or the worker rations may not have provided adequate diets. Furthermore, the synergistic relationship between inadequate diet and infectious disease would serve to amplify the effect of social stratification on the pattern of stress within a population. For instance, malnourished peasants living in crowded conditions would reveal a significantly higher frequency of stress than would the elite who had better access to food and more sanitary living conditions.

Climate change, such as occurred in northern Mesopotamia at the end of the 3rd millennium, would affect the entire population to varying degrees since it is derived from the physical rather than the cultural environment. The climate change, considered abrupt and resulting in a period of marked aridity, would have affected the dry-farming agricultural base. Throughout the long culture history of Tell Leilan one aspect has remained constant: a dry-farming agricultural practice.

Effect of climate change on dry-farming agriculture

Since the northern plains of Syria are crossed with a series of minor ridges (e.g. Jebel Sinjar) and ravines cut into the limestone by seasonal wadis (e.g. Wadi Jarrah), cross country canals or other irrigation schemes would not be permitted except as "enormous engineering projects" (Postgate 1994:11). The geography, therefore, strongly encourages settlement of the region definable by rainfall patterns. In northern Syria, the region of adequate rainfall is geographically definable by isohyets: an *isohyet* defines the minimum amount of rain in millimeters needed to grow a specific crop. For example, barley would need approximately 200 millimeters of winter rainfall and wheat would need about 250

millimeters (Postgate 1994). The isohyets run northeast from modern Aleppo in an arc parallel to the mountains and curve south-east across the Tigris above Ashur (Postgate 1994; Wilkinson 1994) and areas within this region have similar rainfall patterns.

However, as Postgate (1994:13, the author's emphasis) explains, noting base amounts of rainfall for specific crops underestimates the actual rainfall needs of long-term settlement. Rain-fed farming "...requires a locality which can *depend* on adequate rainfall in at least three years out of five..." which, for barley and wheat, translates to approximately 300 millimeters of annual rainfall. Since specific crops are dependent on a base amount of rainfall and dry-farming requires at least a trend of adequate rainfall, a reduction in the average annual rainfall over a substantial period of time would not only directly affect viable agriculture but long-term settlement as well (Figure 2.4).

This scenario is further complicated by cultural factors that could increase long-term settlement vulnerability. In a discussion of the amount of land needed for a settlement based on site size, Wilkinson (1994:499) notes that the increase in the size of Tell Leilan, from the pre-urban period to the urban period, coupled with the increase of the size of surrounding sites, would have put Leilan at its agricultural limit. The author proposes that such settlements would have been "...very vulnerable to famine precipitated by climatic fluctuations...", especially if land restrictions lead to further intensification of agriculture (e.g. denying the cultivation-fallow rotation).

Paleoenvironmental data from Tell Leilan indicate a period of increased aridity and intensified wind turbulence with an elevated dust veil, and have been interpreted to signify a decrease in soil moisture, an increase in aeolian loss of soils and reduced ground visibility. These conditions are central to Weiss' hypothesis (Weiss et al. 1993; Weiss 1996) in that they would have significantly lowered agricultural productivity since an increase in aridity would alter the isohyet patterns thereby affecting crop production.

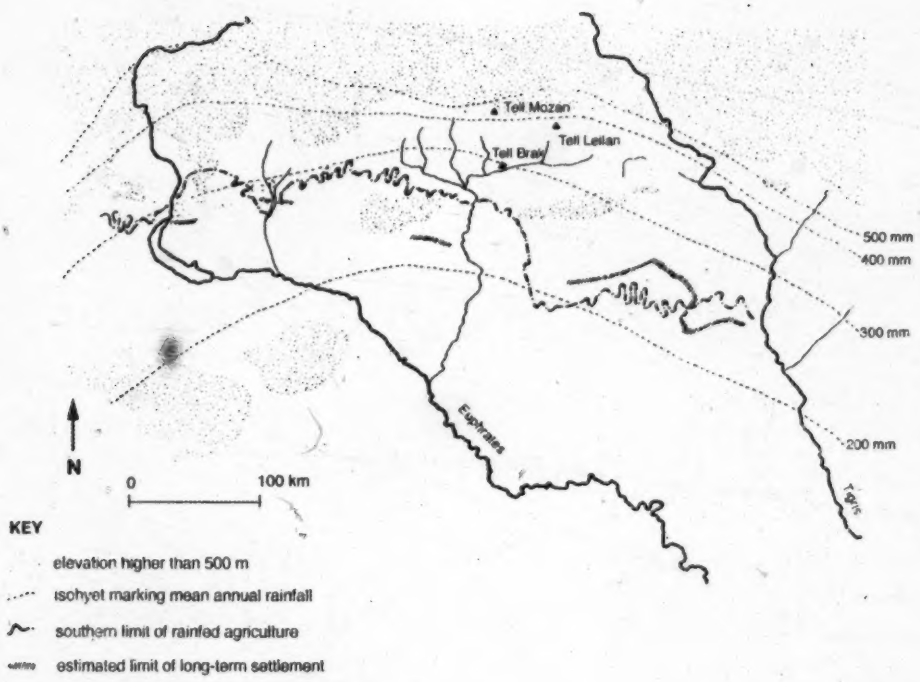

Figure 2.4: Map showing land elevation above sea-level, rainfall isohyets, the estimated range of long-term settlement and the southern limit of rainfed agriculture (adapted from Wilkinson 1994:485).

Over time, the effect on crop production (e.g. crop failure) would have disabled the dependent northern Mesopotamian cities.

During the late Ninevite V period, this trend toward a more arid climate was not, however, associated with any discernible evidence of disruption to the settlement. Indeed, the subsequent cultural period sees the consolidation of complex state administration. Weiss et al. (1993:997-998; Weiss 1986) propose that land-use strategies developed during the late Ninevite V period "...may have presented adaptive advantages by facilitating maximum agricultural production under increased variability of rainfall...". Furthermore, the Akkadians used waterway maintenance (i.e. channelization) as a means

of adapting to the continued trend of increased aridity during the Akkadian imperial period. In considering such a history of adaptive strategies for dealing with an increasingly arid climate, why then would the climate change at ~2200 BC have devastated the society at Tell Leilan?

The social impact of climate change in northern Mesopotamia

Although the effect of climate change would have fundamentally affected the dry-farming tradition of northern Mesopotamia, it may not have been the direct cause of the collapse of the society and abandonment of the site. In discussing the relationship between climate change and ancient societies, Rosen (1995) addresses the issue of environmental change in the Levant during the Early Bronze Age and its impact on the agricultural economy. She demonstrates that although the tradition of control of agricultural surplus and storage by an elite may be adaptive for short-term drought through a system of redistribution, it would have been maladaptive for the long-term in that it "...inhibited the typically successful responses to drought on the part of the subsistence farmers..." (Rosen 1995:27). The centralized system simply could not handle the trend toward a drier climatic regime and/or one of long-term duration.

The initial effect of climate change at Tell Leilan would have been a substantial decrease in agricultural yield. This decrease may have translated into a blow to the livelihood of the peasant farmer while simultaneously lessening agricultural surplus for the elite at Tell Leilan. In response, the elite classes may have responded in ways that had been successful in the past but in this environmental situation were maladaptive. By not successfully adapting to the new climatic conditions, Tell Leilan, as with other northern settlements, lost its subsistence base. It is at this point that various secondary factors (e.g. famine) would inevitably lead to the collapse of the society.

To summarize, the climate change would not only have directly affected the main subsistence base of the society, but to a degree that would have affected the entire society. This environmental change may also have been compounded by social responses that at

one time were adaptive with short-term droughts but were consequently maladaptive with a more abrupt or long-term climatic change. The abandonment of the site is evidence enough of the severity of the effect on the social system at Tell Leilan at the end of the third millennium BC, but can this be discernible in a stress profile compiled through the analysis of enamel defects as indicators of non-specific stress from other specific forms of stress (e.g. infectious disease, dietary inadequacy)?

The health impact of climate change at Tell Leilan

Due to the synergistic relationship between dietary inadequacy and infectious disease, distinguishing between the two through an analysis of *non-specific* markers of stress is not possible. Furthermore, the secondary effects of environmental change may have increased the pathogen load in a number of ways and would also be indistinguishable from direct effects (e.g. famine): for example, members of the rural population may have moved to the city in search of food which would have increased the population density of the lower classes allowing for increased disease transmission as well as increased stress on food reserves. Cultural changes such as increased population size and density could affect the frequency of enamel defects (i.e. stress), and increased social stratification could focus the pattern of stress onto specific segments of the society (e.g. peasant classes). However, it is not until the new climatic conditions leading to the abandonment of the site (i.e. the collapse of the society) that all members would be affected, albeit possibly to varying degrees, with the loss of the subsistence base. With all or nearly all of the society being affected by the climatic change and its secondary effects (be it maladaptive social response or synergism between malnutrition and disease), one would expect to see not only an increase in the frequency of individuals affected but also in the distribution of stress within the society during the period preceding the "Habur hiatus 1" as compared to previous periods or the subsequent reoccupation of the site.

Conclusion

In this chapter, the culture history of the multi-period site of Tell Leilan has been reviewed. Through this review, three themes of change have become evident; change in settlement size and density, social complexity, and climatic conditions. All of these themes could put stress on the health of the population and result in defective dental enamel. Changes in settlement size and density could alter the frequency of defects with the potential of disease transmission and through the synergistic relationship of malnutrition and disease, affect dietary adequacy. Social stratification could alter the pattern of stress by targeting specific segments of the society and may even amplify defect frequency. Changes in climatic conditions could have devastated the subsistence base of the dry-farming society that would have affected the entire population to some degree. Maladaptive social responses to climate change would have initiated a series of secondary effects of the climate change and would have ultimately led to the collapse of the society and abandonment of the site. It has been concluded that not only a higher frequency of individuals but also more segments of the society would be affected in the period preceding abandonment of the site as compared to previous or subsequent periods.

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Chapter 3:

Developmental enamel defects as indicators of non-specific stress: current synthesis and potential problems

Introduction

Clinical and experimental evidence of the link between developmental enamel defects as indicators of non-specific systemic stress, and the epidemiological application of analyzing defects at the community level to assess demographic and socioeconomic patterns of disease, have made the analysis of enamel defects popular in anthropological studies. Furthermore, the durability and indelible record of dental enamel, and the apparent ability to determine the "age of occurrence" of the defect have made dental indicators of stress more popular than skeletal markers for assessing stress profiles reflecting cultural patterns of change (Goodman and Armelagos 1985; Goodman et al. 1984a; Huss-Ashmore et al. 1982; Rose et al. 1985).

This popularity is evident in the numerous studies attempting to identify the effects of culture change on health (e.g. the transition to agriculture) as well as possible cultural trends (e.g. weaning) (see Goodman and Rose, 1990, 1991; Skinner and Goodman 1992, for reviews of past studies). Anthropological studies of developmental enamel defects have not been limited to paleopathological investigations of past cultures, but have also been employed in paleoanthropology (Bermudez de Castro and Perez 1995; Molnar and Molnar 1985; Olgivie et al. 1989; Robinson 1956; White 1978) and in alloprimate studies (Guatelli-Steinberg and Lukacs 1998; Miles and Grigson 1990; Schuman and Sognnaes 1956; Vitzthum and Wikander 1988).

Although the validity of enamel defects as being developmental and non-specific in nature has been proven through clinical and experimental studies, the anthropological application of defect analysis has been under recent scrutiny. Specific methods of defect analysis,

unique to anthropology, have been created through the study of archaeological material. However, a realization of the intra- and inter-tooth variability in defect frequency and expression, and a reappraisal of enamel development at the histological level have forced anthropologists to reassess these methods. This chapter will provide a synthesis of what is known about developmental enamel defects - enamel hypoplasia (e.g. Figure 3.4) and enamel hypocalcification (e.g. Figure 3.5) - and will then address the current methodological problems under question. To begin with, a review of enamel development at the microstructural and histological levels will be necessary.

Dental enamel development

Dental development is a long process beginning before birth and continuing through late childhood and early adolescence. In terms of embryological dental development, Rugg-Gunn (1993:15) explains that the first signs of dental development occur at approximately 28 days of intra-uterine life "...with the appearance of horseshoe-shaped epithelial thickenings..." called *primary epithelial bands* at the lateral borders of the oral cavity where the maxillary and mandibular processes meet. At 37 days, these bands enclose the underlying *mesenchyme*, the embryonic mesoderm or middle primary germ layer of the embryo (Thomas 1989) and, subsequently, the inner portion of the epithelial bands become the dental lamina. The *dental lamina* are a u-shaped growth of epithelium found in both the maxillary and mandibular regions that form enamel organs which later become the tooth germs of the deciduous teeth and permanent molars (Thomas 1989). Between the fourth and tenth months intra-uterine, the permanent incisors, canines and premolars begin to develop out of the tooth germs of their deciduous predecessors.

Tooth germs develop through three stages: the bud stage, the cap stage and the bell stage. There is a close interaction between the epithelial cells forming the bud, cap and bell, and the underlying mesenchymal cells (Rugg-Gunn 1993). The mesenchymal cells adjacent to the inner surface of the epithelial bell differentiate into *odontoblasts* that later form the dentine. Epithelial cells on the inner surface of the bell form the internal enamel epithelium

that differentiates into ameloblasts. *Ameloblasts* are the enamel forming cells and are ultimately responsible for amelogenesis (enamel formation).

Enamel Formation

Enamel formation is a process of appositional growth that passes through several stages; secretion, mineralization and maturation. The *secretion phase* involves the formation of the organic matrix of enamel. Enamel secretion begins along the dentinoenamel junction at the cusp or incisal tip and proceeds cervically in layers towards the neck of the tooth. Ameloblasts initially secrete a protein matrix, forming the basic structure of the enamel crown and subsequently perform a resorptive and a transportive function (Reith and Cotty 1967) within the mineralization phase.

The *mineralization phase* consists of crystal formation and crystal growth. Human enamel crystals are composed mainly of apatite, a mineral similar to hydroxyapatite, which largely consists of calcium and phosphorous. As Posner (1985) explains, apatite crystals are of poor crystallinity and are, therefore, chemically unstable and receptive to ionic exchange. Through ionic exchange within the crystal lattice, apatite crystals form and grow.

During the *maturation phase*, the organic content (namely water and proteins) decreases and the mineral or crystal component increases, mainly through an increase in crystal size (Osborn 1981). The maturation phase is not complete until after the tooth erupts into the oral cavity and, therefore, involves pre-eruptive and post-eruptive maturation phases. Part of the post-eruptive phase of enamel maturation involves ionic exchange with saliva within the oral cavity (Ten Cate 1994).

The end result of enamel maturation is a highly mineralized composite material: a mineral phase comprising 96% and an organic phase making up the remaining 4% (Ten Cate 1994). The mineral phase of enamel consists of densely packed apatite crystals. The organic phase is mainly made up of tyrosine-rich amelogenin proteins, non-amelogenin

proteins and water (Ten Cate 1994), which forms an intricate network between the apatite crystals (Eisenmann 1994). Since the formation of the enamel of the tooth crown is a result of appositional growth with secretion being immediately followed by mineralization, tooth cusps or tips will be undergoing mineralization while more cervical enamel is being secreted (figure 3.1). The process of appositional growth has been likened to a tree-ring analogy and can be better understood through a review of enamel microstructure and histology.

Enamel Microstructure

The basic unit of dental enamel is the *enamel rod*, which "...owes its existence to a highly organized pattern of crystal orientation..." (Eisenmann 1994:242). Although the literature often cites the enamel prism as the basic unit of the enamel mineral phase, this term was adopted before the optical interference of analyzing thin sections of enamel was understood (Eisenmann 1994). It was believed that the basic units were hexagonal and prism-like, hence the term "enamel prism". However, as Eisenmann (1994:240) states, the basic unit of the mineral phase of enamel "...does not have a regular geometry and does not in any way resemble a prism...", therefore, enamel rod is a more appropriate term.

As Eisenmann (1994) explains, an enamel rod is cylindrical in shape and consists of apatite crystals with their long axis running parallel to the longitudinal axis of the enamel rod. This orientation is most clear for crystals along the central axis of the rod, whereas more peripheral crystals tend to flare laterally. An *interrod* area is one in which crystals have different orientations (i.e. flaring laterally) from those making up the bulk of a rod (i.e. parallel to the long axis). The boundary where rod crystals meet interrod crystals creates a sharp angle that causes a larger space than seen between rod crystals and this larger space translates into a higher organic content. This boundary is known as the *rod sheath*.

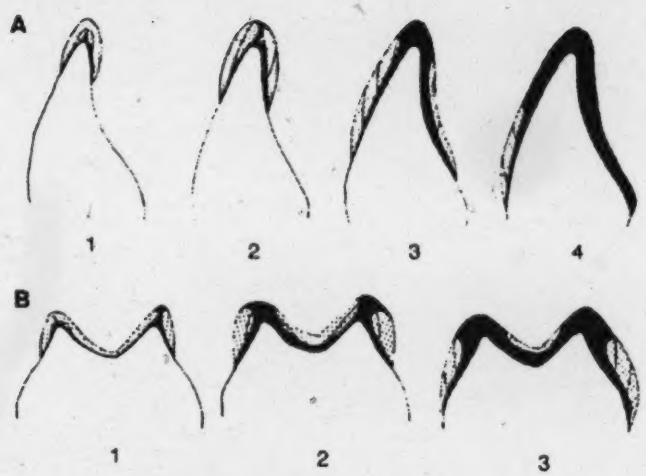

Figure 3.1: Illustration showing the appositional growth of the crown and the progression of enamel secretion (stippled) followed immediately by mineralization (black) (A = an incisor tooth; B = a molar tooth) (adapted from Crabb and Darling 1962:37).

The interrod region located cervical to an enamel rod is not separated by a rod sheath because the crystals are confluent with the rod crystals (Eisenmann 1994). This is evident in a longitudinal section where the orientation of laterally flaring crystals in the most cervical region is nearly perpendicular to the rod (Figure 3.2). Although in cross section this crystal pattern has been compared to a keyhole configuration (e.g. Skinner and Goodman 1992), Eisenmann (1994) warns that this keyhole analogy does not account for certain variations in structural arrangement nor with the secretion pattern of the Tomes processes (a *Tomes process* is a conical process that develops at the apical end of an ameloblast during enamel secretion; Avery 1992). In place of the keyhole analogy, it is considered best to note the specific spatial relationship of the rod-interrod regions cervically and the lack of a definable rod sheath. Eisenmann (1994:242) notes that it is the "...intricate pattern of variations in crystal orientation..." that defines the complexity of enamel.

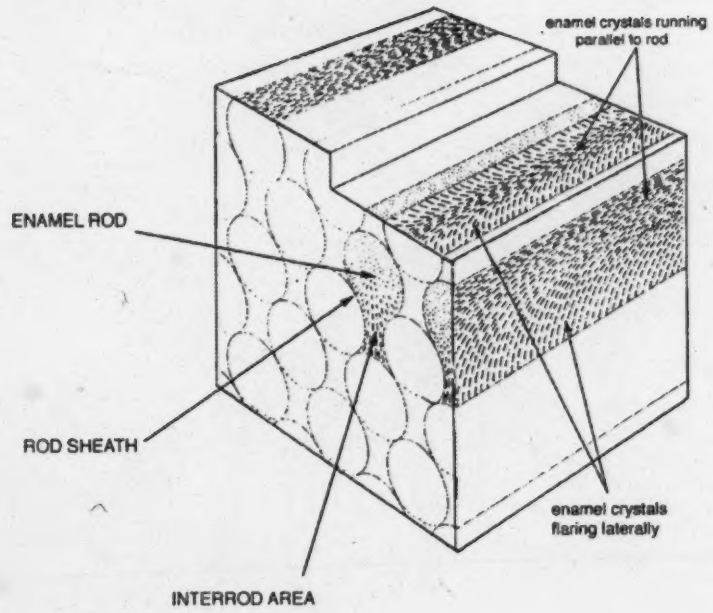

Figure 3.2: Diagram of enamel microstructure identifying the enamel rod, the rod sheath and the interrod area. Note the orientation of the enamel prisms (i.e. running parallel or flaring laterally) and their relationship to the identified features (adapted from Eisenmann 1994:242).

The structure and configuration of the enamel crystals are the responsibility of the ameloblasts and their Tomes processes. During active secretion, the interrod enamel forms first, creating a pit where, in mature enamel, the rod enamel should be. The pits in the immature enamel are occupied by a Tomes process which secretes rod enamel. Based on current knowledge of enamel formation, each ameloblast is responsible for one rod and a portion of the surrounding interrod region (Eisenmann 1994).

Eisenmann (1994) describes the complex interrelationships of enamel rods. The rods are arranged circumferentially in rows around the long axis of the tooth. Each row is situated perpendicular to the surface of the dentine inclining slightly outward toward the cusp. In

the cusp region, the rods have a small radius and run more vertically, whereas in the cervical region, the rods run horizontally for the most part with a few tilted apically. Two other patterns are superimposed on the enamel structure which further complicate the analysis of interrod relationships. Firstly, along the transverse plane of the tooth, each rod has an undulating course from the dentinoenamel junction to the enamel surface, bending medially and laterally. Secondly, "...although the rods in a row run in similar directions, a change in direction of about 2 degrees occurs between successive rows..." (Eisenmann 1994:244).

To summarize, enamel microstructure relies on apatite crystal orientation and the interrelationship of enamel rods. Through analysis of crystal orientation, three features are discernible; the enamel rod (the basic unit of enamel structure), the interrod areas, and the rod sheath. The interrelationship of enamel rods is complex and it is this complexity that is responsible for the unique microstructure of dental enamel and further defines its histological features.

Histological Features of Dental Enamel

The apatite crystals found in dental enamel are highly organized and specifically oriented and it is the differences in crystal orientation that is responsible for much of the histological structure of enamel (Ten Cate 1994). Eisenmann (1994:246) describes different histological structures of dental enamel in terms of apatite crystal orientation; the striae of Retzius, cross striations and the perikymata. The *striae of Retzius* are incremental growth lines "...reflecting successive enamel-forming fronts...". In longitudinal section the striae of Retzius are seen as a series of dark bands, whereas in cross section they are seen as concentric rings. The shape of the tooth (e.g. an incisor versus a molar) is maintained by the striae of Retzius during enamel matrix secretion (Skinner and Goodman 1992). The striae of Retzius are most prominent in the permanent dentition, less prominent in the deciduous dentition, and rare in pre-natal enamel (Eisenmann 1994). Any physiological disturbance occurring during amelogenesis can

result in an accentuation of these incremental lines; for example, the neo-natal line is an enlarged stria of Retzius and reflects physiological changes occurring at birth (Eisenmann 1994). The structural basis of the striae of Retzius has been suggested to be a "...constriction of the Tomes processes associated with a corresponding increase in the secretory face forming interrod enamel...", resulting in altered enamel along these lines (Eisenmann 1994:246).

Cross striations are periodic bands occurring at approximately 4 μm intervals across the enamel rods. Since human enamel is considered to grow at a rate of approximately 4 micrometers a day, cross striations are considered to indicate a daily or circadian variation in ameloblast secretion (Boyde 1979). This chronology is further developed if the striae of Retzius are considered to represent a weekly rhythm of ameloblast secretion (Eisenmann 1994). However, the structural basis of the cross striations is not yet fully clear and at present remains somewhat ambiguous (Eisenmann 1994; Gohdo 1982; Skinner and Goodman 1992).

The striae of Retzius extend from the dentinoenamel junction to the outer surface of enamel and terminate with the perikymata (figure 3.3). The *perikymata* are the shallow ridges and furrows found between the striae of Retzius on the enamel surface, and run circumferentially, in horizontal lines (Eisenmann 1994). If the striae of Retzius are considered to represent a weekly rhythm of ameloblast secretion, and each perikyma furrow represents one stria of Retzius (Risnes 1984), then perikymata furrows have been considered to coincide with one week of enamel development and may be studied in place of the striae of Retzius (Dean 1987; Hillson 1993; Risnes 1985a, 1985b).

Dental enamel development is a complex process with intricate detail. Any disruption in normal amelogenesis can result in defective enamel: a disruption during the secretion phase will result in a reduction in the quantity of enamel (i.e. hypoplasia) whereas a disturbance during mineralization results in enamel of compromised quality (i.e. hypocalcification).

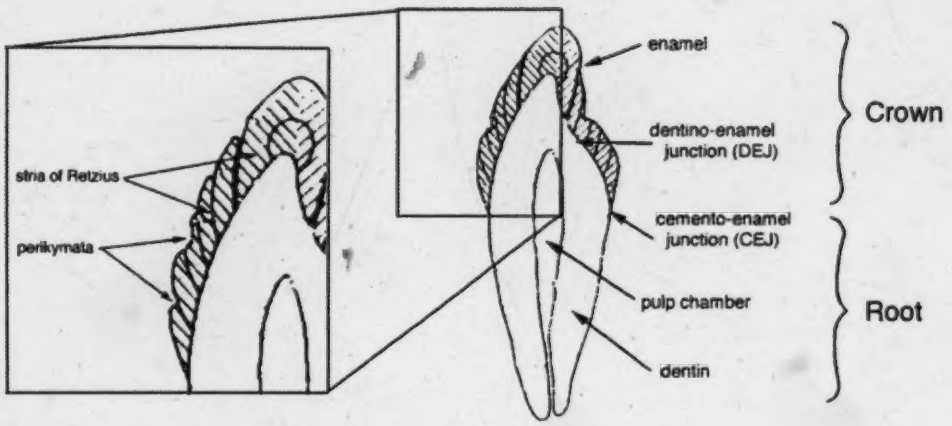

Figure 3.3: Illustration of the anatomy of a tooth. Inset shows histological features discussed in the text (adapted from Goodman and Rose 1991:280; Rose et al. 1985).

Developmental defects of enamel

Three broad etiological categories have been defined that may result in defective dental enamel and which may be identified by the prevalence and distribution of defects within the dentition: hereditary conditions, local trauma and systemic metabolic insults (Goodman and Rose 1990; Shawashy and Yaeger 1986; Suckling 1989). Defects stemming from hereditary or genetic conditions, such as amelogenesis imperfecta (Phakey et al. 1995; Pindborg 1982), can be noted at birth. These defects tend to incorporate the entire tooth crown (Winter and Brook 1975) and will most likely affect all the teeth with the greatest severity (Goodman and Rose 1990; Stewart and Poole 1982; Weimann et al 1945). Goodman and Rose (1990) note that prehistoric cases of enamel defects which are genetic in origin are rare (e.g. Cook 1980). Defects resulting from localized trauma are also considered rare (e.g. Goodman and Rose 1990) and would be limited to a specific part of the dentition (Andreasen et al. 1971; Noren et al. 1993; Rasmussen et al. 1992; Skinner and Hung 1989; Stewart et al 1982; Goodman and Rose 1990). Enamel defects may also be the result of systemic metabolic stress and are referred to as "developmental" enamel defects. As Goodman and Armelagos (1985b:479) explain, a systemic insult will affect

both the right and left antimeres of a tooth type and any teeth developing at the same time and, more specifically, "...the location of the defect on these teeth will reflect the relative completeness of the crown development at the time of the stress..." (Pindborg 1970, 1982; Sarnat and Schour 1941; Weinmann et al 1945). The majority of anthropological analyses of dental enamel defects have focused on defects which are developmental in nature and are, therefore, considered to be the result of a systemic metabolic insult (i.e. physiological stress).

Developmental defects of enamel are defined as reflecting compromised or abnormal enamel structure and are usually classified into two categories: hypoplasia and hypocalcification (FDI 1982; Suckling et al. 1989; Weinmann et al. 1945). Each category reflects a disturbance during a specific phase of enamel development and a unique process creating the defect. Enamel defects may be seen by the naked-eye or under low magnification (i.e. hypoplastic or hypocalcified enamel) and may be referred to as *surface defects*. Defective enamel seen in thin-sections under higher magnification are termed histological defects or *microdefects*. Microdefects, such as "pathological striae" (Gustafson 1959; Gustafson and Gustafson 1967) or "Wilson bands" (Rose 1977, 1979; Goodman and Rose 1990) have been described as accentuated striae of Retzius (Wilson and Shrouf 1970) and can be developmental in origin. Although clinical case studies have linked microdefects at the histological level with systemic stress (e.g. Dahloff et al. 1994), many anthropological investigations attempting to link histological microdefects to surface counterparts (e.g. Goodman and Rose 1990) have not been successful. However, as Hillson and Bond (1997:101) note, "...the appositional zone of a tooth crown hides a considerable proportion of enamel layers..." thereby obscuring crown surface evidence of a stress episode (i.e. a hypoplastic defect).

Enamel Hypoplasia

Enamel hypoplasia is defined as a deficiency in enamel formation resulting in a reduction in the quantity of enamel (Goodman *et al.* 1984; Langlais and Miller 1992).

Histologically, the deficiency in enamel formation is a result of disruption in the secretion of enamel matrix by the ameloblasts during amelogenesis (enamel development) (Boyde 1970; Goodman *et al.* 1980). The most common presentation of "...acquired enamel hypoplasia is a horizontal groove located at the position of the tooth crown corresponding to its stage of development at the time of injury..." (Seow 1991:444) and is usually referred to as linear enamel hypoplasia (LEH) (Figure 3.4). Enamel hypoplasia may also manifest itself as pits or large patches of malformed or absent enamel (Ensor and Irish 1995; Goodman and Rose 1990; Langlais and Miller 1992). The differential expression of enamel hypoplasia (e.g. pits, grooves or focal loss of enamel) is unclear and may reflect different causal factors, the insult occurring during different phases of enamel development (Langlais and Miller 1992), different crown geometry within and between tooth types (Hillson and Bond 1997), or possibly the severity or duration of stress (Langlais and Miller 1992).

The etiology of enamel hypoplasia is not always clear and, in most anthropological cases, assigning a specific cause to a hypoplastic defect is impossible. Although it is known that the lesions represent a disruption in enamel formation due to physiological stress, determining the type or cause of the stress may be futile. Broad clinical classifications of systemic factors affecting amelogenesis include; birth trauma, infections, nutritional disorders, metabolic diseases and chemicals (Seow 1991). Although etiological studies have concluded that enamel hypoplasia results from specific physiological disturbances, the inability to distinguish hypoplastic defects resulting from dietary inadequacy (i.e. a diet insufficient in essential vitamins, minerals, protein and/or calories) from those of infectious disease promote consideration of these defects as indicators of general environmental stress rather than markers of specific diseases or metabolic conditions (Cook and Buikstra 1979; Corrucini et al. 1985; Ensor and Irish 1995; Goodman et al. 1980; Kreshover 1960; Rose *et al.* 1985).

Anthropological inquiries into health in antiquity maintain two general types of stress; inadequate diet (i.e. malnutrition) and infectious disease, and the synergistic interplay

Figure 3.4: Linear enamel hypoplasia (LEH) on the labial surface of a mandibular incisor.

between the two. Numerous researchers have used the presence of enamel hypoplasia to represent dietary stress (i.e. malnutrition or famine) (e.g. Goodman *et al.* 1980; Goodman 1991; Infante & Gillespie 1976; Jelliffe & Jelliffe 1971; May *et al.* 1993; Nikiforuk and Fraser 1981; Sawyer & Nwohu 1985; Smith and Perez 1986; see also Goodman and Rose 1991 for a review of dietary interpretations from enamel hypoplasia). Although a nutritional deficiency may result in a disruption of amelogenesis, it is not always the primary causal factor. Severe infections during amelogenesis, as well as the fevers often associated with infection, may result in hypoplastic dentition (Mason and Roberts 1995; Seow 1991, Smith and Miller 1979, Stodder 1997; Sweeney *et al.* 1969). Furthermore, the etiological basis for hypoplastic dentition may be the result of the synergistic relationship between inadequate diet and infectious disease (Goodman 1991; Skinner and Goodman 1992). For example, due to malnutrition, the body's immune system may be

more susceptible to disease. Similarly, a chronic illness may disrupt an individual's normal nutritional requirements.

Anthropological analysis of enamel hypoplasia commonly scores the defects by type (grooves, pits or focal loss of enamel) and location on the tooth. The location is normally measured on the labial/buccal surface from the midpoint of the cemento-enamel-junction (CEJ) to the most occlusal portion of the defect (Buikstra and Ubelaker 1994). Measurements are taken from the CEJ rather than the incisal edge or cusp tip due to potential obliteration of these features by dental attrition, a common concern with archaeological populations. This measurement is then used to calculate the "age of occurrence" of the defect through the use of standard tables (e.g. Rose *et al.* 1985; Goodman and Rose 1990) based on the dental development chronology of Massler and colleagues (1941).

Enamel hypocalcification

Hypocalcified enamel is defined as enamel of reduced quality which is reflected in a color change and/or increased opaqueness and decreased translucency (Suckling et al. 1989). The reduction in quality is a result of a disruption during the mineralization phase of amelogenesis. This disruption results in a compromised microstructure which may reveal an altered enamel prism relationship and a potential increase in the organic component of enamel (Fejerskov et al. 1988). Hypocalcification, also referred to as enamel opacities or hypomineralization (Seow 1997), may be localized to a single tooth type, affecting part or the entire tooth crown, or incorporate all of the teeth (Small and Murray 1978), and may have vague definition (diffuse opacities) or clear boundaries (demarcated opacities) (FDI 1982) (Figure 3.5). Several different terms have been used in clinical and epidemiological literature in reference to defects in the quality of dental enamel; mottled enamel, dental fluorosis, development opacities, internal enamel hypoplasia and enamel hypocalcification (Small and Murray 1978).

Figure 3.5: Maxillary central incisors with diffuse opacities indicative of enamel hypocalcification (from Croll 1991:19, figure 1-13).

As with hypoplastic defects, hypocalcified enamel may be the result of numerous physiological causes broadly classified as genetic, traumatic or systemic in nature. However, the etiological interpretation of hypocalcified enamel has been heavily biased by clinical and epidemiological investigations of dental fluorosis. Most studies of enamel opacities rely heavily on the information gained in the 1930's and 1940's with Dean's pioneering work on "mottled enamel" and his classification system based on defect severity (Dean 1934). Although the progressive pattern of severity of hypocalcified enamel with increased fluoride incorporation is well known for dental fluorosis, the various causes of non-fluorotic defects of enamel are not obvious by defect appearance and are often simply defined as being idiopathic or "non-fluorotic" in origin (Pindborg 1982; Small and Murray 1978). Indeed, Small and Murray (1978) have identified ninety-seven etiological factors other than fluorosis that may result in hypopcalcified enamel.

The DDE index has been recommended for use in anthropological studies of enamel defects (Goodman and Rose 1990; Hillson 1996) and the current synthesis of anthropological methods, *Standards for Data Collection from Human Skeletal Remains* (Buikstra and Ubelaker 1994), promotes a recording system based largely on the criteria and classification of the DDE index: hypoplastic defects are recorded by type and enamel opacities as to color and boundary characteristics. Furthermore, the location of the

hypocalcified defect is measured in the same manner as hypoplastic defects (i.e. from the midpoint of the CEJ to the most incisal/occlusal aspect of the defect on the labial surface of the tooth).

In comparison to the analysis of hypoplasia, there have been few anthropological cases incorporating enamel hypocalcification as a measure of non-specific systemic stress in archaeological populations (Hillson 1996). Most cases incorporate enamel opacities along with hypoplastic lesions as enamel defects (e.g. Blakely and Armelagos 1985; Duray 1996). However, a few studies have focused on hypocalcification: for example, Lukacs and colleagues (1985) discuss enamel opacities (along with staining and pitting) as a possible result of fluorosis from the Neolithic/Chalcolithic site of Mehrgarh, Pakistan.

Developmental enamel defects as indicators of non-specific stress

The link between systemic stress during amelogenesis and the presence of enamel defects has been identified by experimental and clinical research. These studies also provide evidence that developmental defects of enamel are non-specific in nature and related to a wide variety of stressors (Kreshover 1960). Goodman and Armelagos (1985) provide a review of past experimental studies using rats and different stressors: fever (Kreshover and Clough 1953), infectious agents (Kreshover and Clough 1953; Kreshover et al. 1953), under and over nutrition (Becks and Furnata 1941; Paynter and Grainger 1956; Walbach and Howe 1933) and hormonal changes (Baume et al. 1933; Schour and Van Dyke 1932; Schour et al. 1937; see also Cutress and Suckling 1982; Jontell and Linde 1986; Pindborg 1982 for comprehensive reviews of past clinical or experimental studies). More recent clinical and experimental studies (e.g. Dahllof et al 1994; Giunta 1998; Robinson et al. 1992) also maintain the link between systemic stress during amelogenesis and defective enamel, and the non-specific nature of the defect.

Epidemiological studies have also documented a general association between physiological or metabolic stress, such as malnutrition and/or disease, and enamel defects at the community level, and have related the stress within demographic (e.g. age and sex) and socioeconomic (e.g. poverty) contexts. For example, individuals from developed countries tend to have lower rates of enamel defects than individuals from underdeveloped areas. Cutress and Suckling (1982; Goodman and Rose 1991) note that less than 10% of individuals from developed industrialized countries exhibit one or more enamel defect, whereas enamel hypoplasia is relatively common in children in Third Word countries (Anderson and Stevenson 1930; Baume and Meyer 1966; Jelliffe and Jelliffe 1971; Mollar et al. 1972; Enwonwu 1973; Schamschula et al. 1980; Sawyer and Nwoku 1985; Goodman et al. 1987; Sweeney et al. 1971). A more recent study has linked a lower prevalence of defects with nutritional supplementation (May et al . 1993).

With the positive correlation between systemic stress and enamel defect formation from clinical, experimental and epidemiological studies, anthropologists soon applied such analysis to archaeological samples to assess changes in health and stress patterns. Hillson (1996) notes three general themes in anthropology for the interpretation of enamel defects as indicators of non-specific stress. The first of these is the transition from hunter-gatherer subsistence to agriculture. Indeed, Goodman and Armelagos (1985; Goodman 1991) note that fifteen of the nineteen regional paleopathological studies in the monograph, *Paleopathology at the Origins of Agriculture* (Cohen and Armelagos 1984), contained data on enamel hypoplasia and revealed changes in the frequencies of hypoplastic lesions with the transition to agriculture. Stressors considered associated with this transition are the risk of seasonal food shortages with the reliance on one food source and a potential increase in disease transmission with sedentarism (Hillson 1996). Second, researchers have used defect frequency to determine the average age at weaning in a given population (e.g. Blakely et al. 1994; Corruccini et al. 1985; Hillson 1979; Moggi-Cecchi et al. 1994, for a review of research into weaning in past populations see Katzenberg et al. 1996). The underlying assumption is that a stress episode would be evident at weaning with a new exposure to disease through the loss of the natural antibodies of breast milk

and a further loss of the nutritional buffer of breast feeding (Hillson 1996) and that this stress would be noted in a population as a chronological pattern. Third, changes in the frequency of enamel defects have been investigated to assess the health consequences of contact and the arrival of Europeans in the New World (e.g. Hutchinson and Larsen 1990; Santos and Coimbra 1999; Ubelaker 1994). The hypothesis is that with the Europeans came new pathogens previously unknown to the indigenous populations of the New World and that the newly arrived Europeans would have faced dietary hardships, and that these cultural changes would be reflected in higher rates of enamel defects (Hillson 1996).

Anthropological studies interpret enamel defects (i.e. hypoplastic lesions) as non-specific indicators of stress (Goodman et al. 1988; Goodman and Rose 1990; Hillson 1996) and this assumption is the basis for the above mentioned models of culture change and patterning. Although the validity of this assumption is solidly grounded in clinical and experimental evidence (Kreshover 1940), the legitimacy of the application of defect analysis in anthropological studies has been questioned. Anthropological analysis most often includes a selection of specific tooth types, identification of defect type (i.e. hypoplastic pits or grooves), determination of defect frequency and calculation of peak age of stress. From these analyses, a stress profile may be ascertained and provide insight into the interaction between culture and health (e.g. transition to agriculture or contact) and, through the mean age of defect occurrence, the identification of possible cultural patterns of stress (e.g. weaning). However, new methodological concerns have instigated discussion and debate regarding a standard methodology for anthropological investigations of enamel defects as indicators of non-specific stress (e.g. Hillson and Bond 1997).

Methodological concerns with the analysis of enamel defects

In a discussion of the history and development of an epidemiological standard criteria for identifying and classifying developmental enamel defects, Clarkson and O'Mullane (1989) explain that over the past half century various indices for classifying enamel defects have

been proposed and may be divided into two main groups; specific fluorosis indices (Dean 1934; Dean et al. 1942; Horowitz et al. 1984; Thylstrup and Fejerskov 1978) which are based on etiological factors, and descriptive indices (e.g. Al-Aousi et al. 1975; Clarkson and O'Mullane 1989; FDI 1982; Jackson et al. 1975; Losee et al. 1961; Murray and Shaw 1979; Smith 1983; Suckling et al. 1976; Young 1973) which encompass all types of enamel defects and rely on a definition of type rather than causal factors (Clarkson 1989). More recently, the Federation Dentaire Internationale (1982) proposed the Developmental Defects of Enamel index (DDE index) which is based on defect description rather than etiology to allow for greater ease and comparability between studies. Despite subsequent modifications (Clarkson and O'Mullane 1989; FDI 1992), the general format of the DDE index has been widely accepted in epidemiological as well as anthropological studies (Hillson 1996).

In attempts to apply epidemiological indices of developmental enamel defects to archaeological dental samples two main problems arise. First, epidemiology deals with living communities whereas anthropology investigates past populations from archaeological contexts. Demographic and socioeconomic data and/or medical histories are not directly available for etiological correlation. The anthropologist must rely on skeletal indications of age, sex and stature for demographic aspects and on archaeologically derived cultural contexts to shed light on potential socioeconomic influences. Secondly, taphonomic processes within the burial environment can limit analysis through the recovery of incomplete dentitions which decrease sample size or by post-mortem alteration of the dental enamel (e.g. staining, root etching or chipping). These problems have instigated the development of specific methods of analysis unique to anthropology, primarily with (1) a main focus on enamel hypoplasia rather than hypocalcification, (2) a concern for the selection of specific tooth types for analysis and (3) an emphasis on the calculation of age of defect occurrence.

1. A main focus on enamel hypoplasia rather than hypocalcification

The majority of anthropological analyses of developmental defects have focused solely on enamel hypoplasia as an indicator of non-specific stress rather than including enamel hypocalcification. Although epidemiological indices have long included hypocalcification as an equal indicator of non-specific stress to hypoplasia, anthropologists have tended to exclude this defect category from their analyses. This may be due in part to possible post-mortem alterations from diagenetic changes or altered water content (Hillson 1996). Since enamel opacities are indicative of altered mineralization, ionic exchange within the burial environment could alter the color or translucency of the original opacity or create pseudo-opacities. In addition, through soil acidity, root etching and groundwater, the surface of enamel can be stained, eroded or marred making the identification of enamel opacities difficult if not impossible. Furthermore, "...prolonged dessication..." may enhance or create artificial opacities (Needleman et al. 1991:213); Suckling et al. (1989:226) found hypocalified enamel most often on the cusps and occlusal ridges but when the enamel was dry, defects were "...more widely distributed over the rest of the crown...". In contrast, enamel hypoplasia has been considered virtually free from post-mortem or taphonomic alteration since the early histological analyses of archaeological teeth by Tomes (1892, cited in Rose et al. 1985).

Antemortem conditions can also affect the frequency and distibution of hypocalcified defects. Needleman et al. (1991;213) note the potential of dental calculus to decalcify enamel and produce "white spot" decalcifiaction, mimicking enamel hypocalcification. As Croll (1991:22,26) explains, enamel hypocalcification is a result of disruption during the mineralization phase of amelogenesis and is, therefore, *developmental*. In contrast, enamel decalcification is *acquired* when dental plaque persists on the surface of the tooth crown and the organic acids etch the mineral out of the enamel.

In a discussion of the macroscopic appearance of enamel defects, Suckling and Thurley (1984) note two factors in regards to enamel opacities that may deter their use in anthropological studies. First, although diffuse opacities usually have a symmetrical

distribution in the dentition (i.e. affecting both the right and left antimeres), demarcated opacities are frequently distributed asymmetrically. An asymmetrical distribution of demarcated defects would, by definition, be indicative a traumatic or hereditary etiology rather than developmental (see above, Developmental defects of enamel), and, therefore, may not be included in an analysis of non-specific stress.

Second, the validity of relating the position of a defect (i.e. hypoplasia) on the tooth surface to an age of occurrence is based in clinical studies of the secretion phase of enamel. Timing of the mineralization and maturation phases of enamel formation are not well understood and for this reason it is not possible to assess the age at which a hypocalcified defect was "...formed from the appearance of the defect and its position on the surface of the erupted tooth..." (Suckling and Thurley 1984:358). Since the calculation of the age of occurrence of a defect is central for determining the peak age of stress within a population and for the identification of cultural patterns, the inability to measure age of occurrence with hypopcalcified defects may limit or deter their use in anthropological studies.

2. Selection of tooth type(s) for analysis

Although evidence from clinical and experimental studies indicate that developmental enamel defects are non-specific in regards to a specific etiology and representative of general environmental stress (Kreshover 1960), several researchers have investigated factors other than environmental stress that may influence the presence, expression and distribution of developmental enamel defects. These investigations have found intra- and inter-tooth variability and differential expression of defects in terms of tooth type, tooth surface and tooth area.

As discussed above (see section on Enamel Defects), enamel defects can have a distinct distribution in relation to general etiology: for example, systemic stress will be reflected symmetrically in both the right and left antimere of a tooth type and in those teeth

developing at the same time. Furthermore, the location of the defect should be at the degree of crown development at the time of insult. The pattern of developmental enamel defects within the dentition has long been assumed to be solely dependent on crown formation and that all teeth are equally exposed and responsive to stress, and it is these assumptions that have been the basis for most anthropological analyses (Goodman and Armelagos 1985b). However, numerous studies reveal different defect frequencies within and between tooth types suggesting a differential susceptibility to stress and/or defect formation.

Goodman and Armelagos (1985b) address this issue and review past studies which provided inter-tooth differences in the presence of enamel defects. In a dental analysis of the Hammon-Todd collection, El-Najjar et al. (1978) noted variation in the frequency of individuals with one or more hypoplastic defects per tooth type and found the greatest frequency in the anterior teeth, followed by the premolars, then molars. Black (1979) found similar results in his study of a prehistoric cemetery from southwestern Missouri: a significantly higher number of hypoplastic lesions in the canines than the premolars or molars. Cutress and Suckling (1982) analyzed the data from five epidemiological studies (Murray and Shaw 1979; Suckling et al. 1976; Richards et al. 1967; Young 1973; Smith 1979) and found the frequency of enamel defects differed by tooth type: maxillary central incisors were the most hypoplastic, then the maxillary and mandibular canines, followed by the mandibular lateral incisors and the maxillary and mandibular second molars as the least hypoplastic. More recently, Moggi-Cecchi et al. (1994) maintained the assertion that anterior teeth are more hypoplastic than are posterior teeth and that the maxillary canine exhibited the most hypoplasia.

In noting that variations in defect frequencies between tooth types are "...common and substantial...", Goodman and Armelagos (1985b:480) analyzed the morphological and chronological distribution and the frequency of enamel hypoplasia by tooth type. Testing the assumption of "uniformity of response" (i.e. developing tooth crowns will be equally exposed and responsive to stress), which underlies the dental literature as well as studies

using enamel defects as indicators of non-specific stress, Goodman and Armelagos (1985b) found extensive variation in the frequency of defects by tooth type. Furthermore, the authors found intratooth variation, whereby defects were not randomly distributed but rather tended to be found in the middle third of the crown for all tooth types. They conclude that factors other than time of crown development control the occurrence of defects; namely, genetic, microstructural and histological factors of enamel development.

Intratooth variation in the expression of defects has also been noted for microdefects and surface defects of the same tooth (Cook 1981; Rose 1977; Goodman et al. 1980; Hutchinson and Clark 1988). Condon and Rose (1992) found Wilson bands in all regions of the crown but found surface defects (i.e. hypoplasia) limited to the cervical two-thirds. Hillson and Bond (1997) reason that the lack of surface defects in the incisal/occlusal third of the tooth crown is due to the process of appositional growth: incisal or cuspal enamel is built up in layers which results in enamel being hidden under subsequent layers which denies the surface expression of internal histological features.

This evidence of intra- and inter-tooth variation in defect expression elicits methodological concerns for epidemiological and anthropological studies. Goodman and Rose (1990) note that the variability warrants careful consideration to the tooth type(s) selected for study: a tooth type not frequently hypoplastic (e.g. a molar) would present an underestimation of stress in a population whereas a tooth type commonly found to be hypoplastic (e.g. the mandibular canine) would produce a high prevalence of stress in a profile. Studies using different tooth types would, therefore, be incomparable.

Standardization of tooth type selection is one of several issues which need to be resolved in terms of methodology. It now seems clear that due to dental development and microstructure, as well as genetic factors, selection of the tooth type(s) to be analyzed has serious implications for anthropological studies of dental defects. Various researchers have made different recommendations as to tooth type selection (e.g. Ensor and Irish 1995; Goodman et al. 1980; Goodman and Armelagos 1985b; Rose et al. 1985), such as

using the single tooth type(s) that are commonly hypoplastic or, alternatively, multi-tooth types for a better chronological sample. But are these recommendations realistic for an archaeologically derived sample?

Factors affecting an archaeological dental sample

Numerous factors can and do affect the dental sample representing the population under study. First, the recovery of teeth from the burial environment can be hampered by several cultural factors and may result in a decreased dental sample: for example, cremations can destroy dental data entirely, intramural burials can be disturbed in antiquity due to subsequent building phases, and secondary burial practices that relocate skeletal remains may lose teeth. Loss of teeth is in part also a function of tooth type, since single rooted teeth such as incisors and canines are lost more often than are multi-rooted teeth such as the molars. Another factor is simply that low density or small settlement sites would not have the original population necessary to produce a significant cemetery sample.

Furthermore, not all recovered teeth are useful in analysis. A common problem in the analysis of teeth from archaeological populations is dental attrition, which can obliterate part of or the entire tooth crown and any evidence of past stress. Since tooth wear is common in many archaeological populations, especially with older adults (as noted with the use of tooth wear for relative aging) and within certain cultures, the analysis of only unworn teeth would bias results and morbidity curves by excluding older individuals. The potential damage from dental attrition can be lessened, however, if a specified, limited degree of wear is permitted (through the use of standardized wear charts), and if teeth that are commonly heavily worn (e.g. first molars) are not selected.

In sum, although genetic, developmental and/or microstructural factors may dictate the rate and expression of defect formation, any attempts to develop a methodology based on tooth type selection must also consider the reality of an archaeologically derived dental sample. The selection of one or two tooth types based on their higher susceptibility of defect formation may be unrealistic for analysis of teeth from smaller sites or those with

specific cultural practices (e.g. intramural or secondary burial). The standardization of a methodology limited to only those sites with large scorable dental samples would in turn limit the potential information gained through population comparison.

3. Calculating age of occurrence

The traditional method in the anthropological analysis of enamel defects has included determining the age of occurrence of the defect by measuring its position on the labial surface of the tooth crown, from the cemento-enamel junction (CEJ) to the most occlusal aspect of the defect, and then converting this measurement to a dental age through established chronological conversion charts. Through the calculation of a mean age of occurrence, a peak age of stress within a population may be determined and provide insight into cultural trends such as weaning.

Recently, however, this standard method of calculating age of occurrence has been criticized. In attempts to expand this methodology to include consideration of the severity and duration of a stress episode, some researchers wish to develop the method to include measurement of the width and depth of the defect to determine the duration and severity of the stress episode (e.g. Blakely and Armelagos 1985; Ensor and Irish 1995; Hutchinson and Larsen 1988). However, Buikstra and Ubelaker (1994:57) claim that "...such inferences have yet to be firmly grounded in clinical evidence..." and suggest noting the width of a hypoplastic feature, especially in cases of severe defects, but maintain that the distance from the CEJ to the most occlusal aspect of the defect is the "...single most important measurement...".

This claim has been challenged by Ensor and Irish (1995) who argue that the "...analysis of length of time involved in stress episodes may provide a more precise measurement of metabolic insult..." than frequencies of hypoplastic events and advocate the need to measure the width of a hypoplastic defect to determine the duration of the insult. The

authors propose a new method, the Hypoplastic Area Method (Ensor and Irish 1995), to analyze frequency and duration of stress within a population.

Based on a distinction between discrete and continuous hypoplastic disturbances, the Hypoplastic Area method (Ensor and Irish 1995) strives to address the amount of stress resulting in a hypoplastic lesion. A discrete disturbance is considered a single event resulting in a localized hypoplastic lesion and is described as pits or "...thin linear disturbances..." of 0.5 mm or less in width. In contrast, a continuous disturbance is considered a long-term episode representing a "...record of stress without immediate recovery..." and are described as being more than 0.5 mm in width. The assumption is that by comparing measurements of continuous defects, information pertaining to levels of stress in a population, beyond that of stress frequency, may be acertained.

The Hypoplastic Area method has been criticized on several levels. In comparison to a previous methodology incorporating duration of stress (Blakely and Armelagos 1985), Blakely and Armelagos (1997:295) criticize the Hypoplastic Area method as permitting "...the error of counting twice the duration of hypoplasia on different teeth, whose development is simultaneous...". In response, Ensor and Irish (1997:297) deny such a direct comparison of methodologies and state that the "...most obvious difference is that the two methods measure different aspects of [enamel hypoplasia]: individual chronicity and stress duration (theirs) vs. specific tooth and/or individual amount/area (ours)...". However, a separate discussion on crown formation and geometry puts methodologies attempting to determine severity or duration of stress into question.

In a discussion of the relationship of enamel hypoplasia to the pattern of tooth crown growth, Hillson and Bond (1997:89) state that the progressive decrease, from occlusal to cervical, in the spacing between developmental layers (i.e. perikymata) of the tooth crown would influence both defect prominence and width, and that it would be "...difficult to use measurements as a means of estimating the duration of the disturbance causing a particular defect...".

The relationship between crown formation and hypoplasia also has serious implications for the calculation of age of occurrence. Hillson and Bond (1997) discuss the fact that defect type and location on the crown are dependent on not only the progressive decrease in the spacing of developmental layers, but the appositional versus imbricational zones of crown growth as well (Figure 3.6). Therefore, simply measuring the defect on the crown surface would be inadequate.

Hillson (1996) notes five fundamental problems with calculating age of defect occurrence. First, the table assumes a constant growth rate for crown formation that is not supported by the geometry of crown development. Second, it denies the appositional growth of crown formation. Third, despite variation in crown height, the table assumes a single mean crown height for each tooth type. Fourth, the table is based on the dental development standard of Massler and colleagues (1941). Later studies have revealed variation from this standard. And fifth, "...the position of a defect on the crown surface is not simply related to the timing or duration of the growth disruption that caused it..." (Hillson 1996:175).

The fifth point is evident in a study of variability in defect expression within and between tooth types: Goodman and Armelagos (1985a) tested the chronological distribution using half year developmental periods of equal width corresponding to their time of crown development (based on Massler et al. 1941) and analyzed the frequency and distribution of hypoplastic defects. The authors found different high and low frequencies of defects between tooth types at the same chronological half year period and conclude that factors other than time of development govern the occurrence of defects.

The problems with the current method for determining age of occurrence of a defect are acknowledged by some researchers (e.g. Seow 1997). However, the potential severity of these problems is often underestimated. For example, in discussing the need for additional standards of tooth formation, Skinner and Goodman (1992:165) advise researchers to

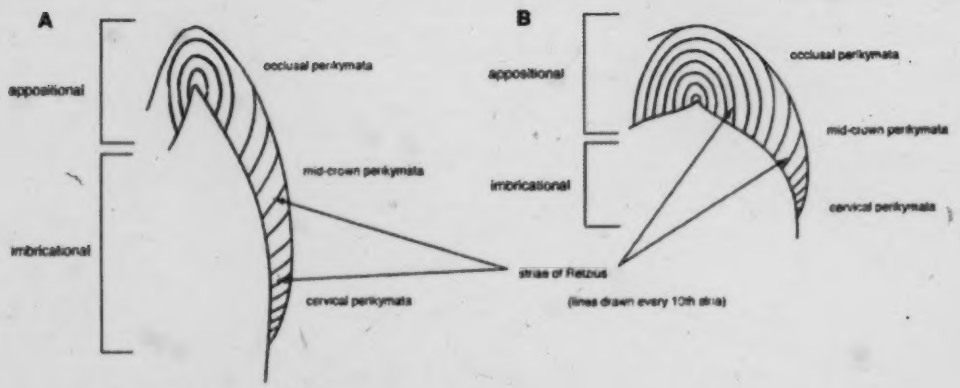

Figure 3.6: Illustration showing the appositional and imbricational regions of the tooth crown, as well as the different areas of perikymata (occlusal, mid-crown and cervical). Note the difference in the ratio of appositional to imbricational growth between the permanent maxillary first incisor (A) and the permanent mandibular second molar (B) (adapted from Hillson and Bond 1997:96).

"...continue to reconstruct age of occurrence from the standard of Massler and colleagues (1941) as long as they retain their raw data for recalibration as new standards come along...". In consideration of the argument by Hillson and Bond (1997; Hillson 1992, 1993, 1996), recalibration of previous measurements may not be simple or direct. As Hillson and Bond (1997:102) note, alleviation of the problems would require a "...concentrated effort on a large number of specimens to define what is normal in terms of crown formation sequence and timing worldwide..." or alternatively, limit the analysis of hypoplasia to the microscopic level with perikymata counts determining the relative timing and sequence of hypoplastic defects.

Conclusion

Are enamel defects valid indicators of non-specific stress? Although clinical and experimental studies have long maintained a link between defect formation and metabolic stress, epidemiological research has expanded this link to include demographic and socio-cultural contexts, such as socioeconomic status. Furthermore, using epidemiological cases as analogies, paleopathological investigations have extrapolated the non-specificity of

defects to represent cultural patterns (e.g. health and subsistence change, weaning). Therefore, the question is not whether or not enamel defects are indicative of non-specific stress, but whether the applications of dental markers of non-specific stress are valid.

In reviewing different issues concerning the anthropological application of developmental enamel defects several points can be made. First, there is a preference for analyzing hypoplastic defects rather than enamel opacities. This bias may be due to potential post-mortem alteration of the defects or the creation of pseudo-opacities, differences in analytical techniques, the possible asymmetrical distribution of demarcated opacities, and/or the inability to calculate age of occurrence. Second, there is concern for the selection of tooth type(s) for analysis. Intra- and intertooth analyses have confirmed variation in defect expression and frequency which, consequently, has serious implications for comparability between studies and calls for a standardization of tooth type(s) selection. Furthermore, the standardization of tooth type selection must also be realistic for archaeologically derived samples. Third, due to a better understanding of crown formation at the microstructural and histological level, the standard method is considered flawed in that it does not account for the formation time of hidden enamel in the incisal and cuspal regions of teeth in the calculation of age of defect occurrence. The standard method, therefore, measures location on the crown surface relative to crown height rather than time of development at which the metabolic insult occurred.

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Chapter 4:
Differential patterns of stress at Tell Leilan (Syria): An intra- and inter-tooth analysis of hypoplastic and hypocalcified enamel defects

Introduction

The analysis of developmental enamel defects as indicators of non-specific stress has been popular in anthropology for determining the effect of culture change on past health. A well-known example is the effect on health of the transition from a hunter-gatherer subsistence to agriculture (see Cohen and Armelagos 1984). More recently, the health impact of culture change has been investigated within numerous contexts; contact and the arrival of the Europeans in the New World (Hutchinson and Larsen 1990; Santos and Coimbra 1999; Ubelaker 1994), health and stress with American slavery (Blakely et al. 1994), the rise of industrialization and urbanization during the 18th and 19th centuries (Wood 1996), and the collapse of past civilizations (e.g. Wright 1997).

Enamel defects resulting from systemic metabolic stress, rather than a genetic or traumatic origin, are referred to as "developmental". Developmental defects of enamel are defined as reflecting compromised or abnormal enamel structure and are usually classified into two categories; hypoplasia and hypocalcification (FDI 1982; Sucklinng et al. 1989; Weinmann et al. 1945). Each category implies a disturbance during a specific phase of enamel development and a unique process creating the defect. Both enamel hypoplasia and hypocalcification are developmental in nature and can be the result of numerous etiological factors (see Seow 1991; Small and Murray 1978) and, due to an inability to differentiate between the numerous different causes, are considered to reflect *non-specific* systemic stress. Therefore, a defect would indicate physiological stress on the individual during the period of tooth crown development (i.e. childhood).

Anthropological studies using developmental enamel defects as indicators of non-specific stress most often focus on enamel hypoplasia and in most cases do not include enamel hypocalcification. Although epidemiological indices have long included hypocalcification as an equal indicator of non-specific stress to hypoplasia, anthropologists have tended to exclude this defect category from their analyses. One possible explanation is the fear of post-mortem alteration in the burial environment mimicking hypocalcified enamel (Hillson 1996). However, the validity of enamel hypocalcification as an indicator of non-specific stress has been doubted at the epidemiological level as well. In a study of enamel defects in relation to socioeconomic and nutritional status, Goodman (1991:284-286) found no significant mean differences in the socioeconomic scores (SES) for individuals with or without enamel opacities as noted with hypoplasia. He concludes that "...the lack of association between enamel hypocalcification and SES or nutritional status suggest that these defects are not related to general conditional of life..." but that the "...degree of association of between hypoplasias and nutritional and socioeconomic status, however, reaffirms the notion that they are related to general conditions of life...".

This chapter will provide a comparison of hypoplastic and hypocalcified defect frequency and distribution within the Tell Leilan permanent dental sample. Since both defect classes are considered to represent non-specific stress, then a similar defect pattern (i.e. stress) should be evident. This study will investigate the possibility that these two defect classes may not represent the same stresses or life conditions.

Health and stress at Tell Leilan

This study will analyze the dental remains from 23 individuals from the site of Tell Leilan. Leilan is a multi-period tell site located on the Khabur plain of northeastern Syria (Figure 4.1). Situated on the bank of the perennial Wadi Jaṛṛah, Tell Leilan rests in the dry-farming region of what is known as the Habur Triangle (Weiss 1991). Encompassing more than 90 hectares, Leilan is "...one of the largest ancient sites in northern Mesopotamia..." (Weiss 1985:6).

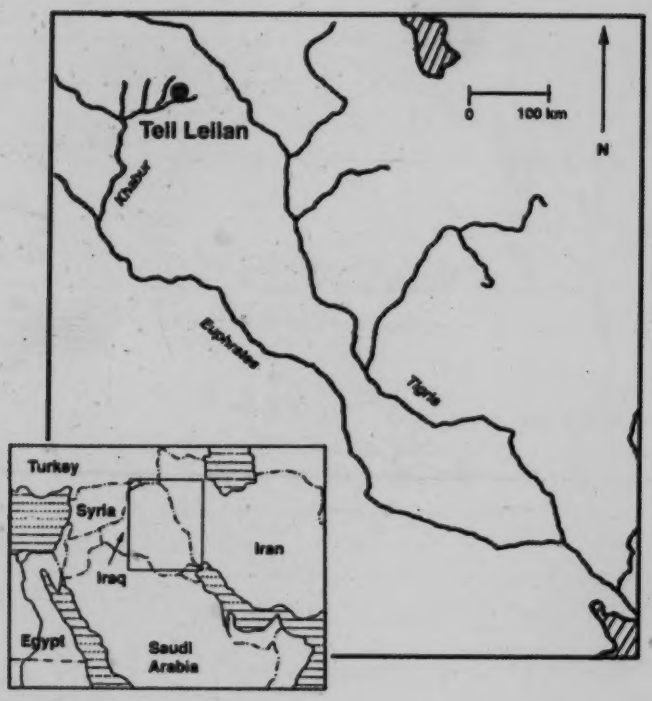

Figure 4.1: Regional map of Mesopotamia identifying Tell Leilan (map adapted from Stein 1994:13; inset from Bernbeck 1995:10).

Historical and archaeological investigations of Tell Leilan have revealed a dynamic culture history spanning several millennia. Six different cultural periods have been identified and reflect times of cultural development and change; Ubaid, Uruk, Ninevite, the Urban period, Akkadian imperialism and the reign of Shamshi-Adad. In reviewing the political, social and economic aspects of each period, three general themes of change are apparent; change in settlement size and density, change in social organization and change in the climate (Table 4.1).

Table 4.1: Chronology of Tell Leilan including general periods, Leilan periods, radiocarbon dates and possible influences on health and stress (based on Bahn 1992; Weiss 1990; Weiss et al. 1993)

General period	Leilan period	Date (BC)	Factors affecting health
Reign of Shamshi-Adad	I	~1990-1728	lower density city, dense distribution of village settlements
Habur hiatus 1	-----	~2200-1900	abandonment; desertification
Akkadian imperialism	IIb	~2300-2200	densely inhabited city; socially stratified; agricultural intensification; climate change
Urban period	IIa	~2400-2300	further increase in social stratification
Ninevite V			
late	IIId	~2600-2400	increase in site size; increased social stratification
middle	IIIb-c	~2900-2600	increase in social stratification?
early	IIIa	~3000-2900	dispersed low density settlements; decreased social stratification
Uruk			
late	IV	~3300-3000	increase in settlement size; increased social stratification
early	V	~4100-3300	
Ubaid (Chalcolithic)	VI	~5500-4100	increased sedentarism and population densities; towns coalesced along rivers
Halaf	-----	~late 6th-early 5th millennium	small settlement size; some increase in social stratification (chiefdoms?)
Hassuna (Neolithic)	-----	~6th millennium	small village-level settlements; minimal social stratification

All three of these changes could influence health and alter the stress profile for the population. For instance, changes in settlement size and density could encourage or discourage the transmission of infectious disease (e.g. Kent and Dunn). An increase in social stratification could target stress onto certain segments of the society, such as malnutrition among the poorer classes, or a decrease could equalize access to resources.

Climatic change could affect the agricultural base and deny adequate nutrition (e.g. famine). Since each of these changes could produce stress on the individual or population in terms of malnutrition, infectious disease, or a combination of both, all three could produce defective dental enamel.

Materials and Methods

The Tell Leilan dental sample consists of 183 permanent teeth, both maxillary and mandibular. Since the dental specimens were recovered from intramural burial contexts (i.e. disturbed and fragmentary) attention was paid to ensure that all teeth belonged to the same individual and that no teeth belonging to the same individual were counted as two individuals. Although each burial was assigned by the excavator to an archaeological period, all of the dental specimens were grouped to compare the defect classes (i.e. hypoplasia and hypocalcification), and as a result, age, sex and status were not considered. Two factors determined the selection of dental specimens to be analyzed. First, teeth with more than slight wear (stage 2 of Smith's methodology, Smith 1984:45) were not included since moderate to severe attrition would destroy any evidence of defects in the incisal/occlusal third of the crown. In addition, other antemortem conditions such as caries or unusual wear patterns were also deleted from the sample. Second, teeth revealing postmortem damage such as chipping, cracking or enamel loss within one of the three areas was also deleted from the sample. This selection process yielded a final dental sample of 153 permanent teeth.

All teeth in the dental sample were scored for calculus using the methodology provided by Buikstra and Ubelaker (1994:56). After the calculus was scored it was removed using a wooden probe to lessen potential damage (e.g. scraping or grooving) to the enamel surface. The teeth were further cleaned by dry brushing to remove any remaining calculus and/or dirt. Acetone applied by a cotton swab or brush was used to remove any consolidant (e.g. polyvinylacetate) that had been applied in the field.

The method involved a visual assessment of type and location of hypoplastic and hypocalcified defects. While only defects seen by the naked eye were recorded, a closer inspection of defects did include the use of a light microscope and a hand lens. Although some researchers (e.g. Buikstra and Ubelaker 1994) suggest analysis of one or two commonly defective tooth types (e.g. maxillary central incisor and mandibular canine), this study employed all permanent tooth types to allow for a larger dental sample and a greater age range (i.e. prenatal to ~15 years). The left antimere of each tooth type was chosen for analysis. When the left antimere was not present or scorable, then the right antimere was used.

The methodology for this study is based on the proposed method for anthropological analyses of enamel defects provided by Buikstra and Ubelaker (1994:56-58), which has been derived from the Developmental Defects of Enamel (DDE) index proposed by the Federation Dentaire Internationale (1982) for use in epidemiological studies. A standard method in anthropological studies has been to calculate a mean peak age of stress: measuring the location of the defect from the cemento-enamel junction (CEJ) to the most occlusal limit of the defect, to provide a measurement which is subsequently converted through standardized tables into an "age of occurrence". This study did not calculate a peak age of stress since the focus of this analysis is to compare defect frequency and distribution between hypoplasia and hypocalcification, not to ascertain age-based cultural patterns.

Defects were recorded as to class of defect, type of defect, and position of defect by surface and by tooth-third location (Table 4.2). A defect is defined as any irregularity or abnormality in the dental enamel. Normal enamel is usually white to cream in color and translucent. The crown surface is smooth with the only ridging (i.e. perikymata) occurring naturally and typically at the microscopic level. Any alteration in the quantity or quality of dental enamel is the result of a systemic or physiologic insult, and results in an enamel defect such as hypoplasia or hypocalcification.

Table 4.2: Dental enamel defects data collection key (based on Buikstra and Ubelaker 1994; FDI 1982)

A. defect	- = not present 0 = unscorable 1 = normal enamel 2 = hypoplastic enamel 3 = hypocalcified enamel (enamel opacity)
B. type	1 = pitting 2 = horizontal grooves (LEH) 3 = focal loss of enamel 4 = diffuse opacity 5 = demarcated opacity
C. surface	1 = lingual 2 = labial/buccal 3 = both surfaces
D. location	1 = cervical third 2 = middle third 3 = incisal/occlusal third 4 = cervical and middle thirds 5 = middle and incisal thirds 6 = entire tooth crown

Enamel hypoplasia is defined as any reduction in the quantity of dental enamel and may appear as a variety of lesions: pitting or horizontal grooves, or as an area of focal loss of enamel. It is the result of a systemic disruption during the secretory phase of amelogenesis (i.e. enamel development). Enamel hypoplasia can appear as a single pit or several pits. Single or multiple hypoplastic pits are recorded under "pits". Horizontal grooves, also known as "linear enamel hypoplasia" (LEH) (Figure 4.2), are also indicative of enamel hypoplasia and may appear on one surface (the lingual or labial/buccal surfaces) or circumscribe the tooth (both surfaces). A focal loss of enamel is defined as an area of enamel reduction that is not a pit or a groove. The etiological and microstructural differences between the different types of hypoplastic lesions are not understood (see Hillson and Bond 1997 for a recent discussion and proposed explanation).

In contrast to hypoplasia, *hypocalcification* results in enamel of lower quality due to a systemic insult during the mineralization phase. Enamel hypocalcification, also referred to

Figure 4.2: Linear enamel hypoplasia (LEH) on the labial surface of a mandibular incisor.

as enamel opacities, alters the normally translucent enamel to an overall opaqueness or patches of opaque or discolored enamel (i.e. staining). The difference in translucency or increased opaqueness reflects the altered microstructure which may be exhibited as a "demarcated" or a "diffuse" opacity. Demarcated opacities, also referred to as discrete, are lesions separated from normal enamel by a definite or well-demarcated boundary. Diffuse opacities are described as "...patchy, irregular, cloudy areas of opacity lacking well-defined margins..." (Federation Dentaire Internationale 1982:160) (Figure 4.3). Discolored enamel can appear as white/cream or brown/yellow. Discoloration will not be considered in this study due to potential postmortem alteration of enamel color in the burial environment.

Figure 4.3: Hypocalcified defects on the labial surface of the maxillary central incisors (from Croll 1991:19, figure 1-13)

The position of a defect was recorded as to presence (on the lingual or labial/buccal surface), and as to surface incorporation (involving the lingual or labial/buccal surface or both surfaces). The mesial and distal surfaces were not considered due to interproximal wear and the lack of full view for teeth still in situ in the maxilla or mandible. The location of enamel defects was further noted as to tooth areas: cervical third, middle third or incisal/occlusal third. If the defect occupied more than one third it was be recorded for all areas it incorporated (e.g. cervical and middle, middle and incisal/occlusal, entire surface). Defect location by tooth third was recorded on the labial/buccal surface only to be comparable with other studies.

Results

A total of 153 permanent teeth were scored for type, surface and location of hypoplastic and hypocalcified defects; 121 (79%) of the teeth were hypoplastic revealing a total of 144 defects and 105 (68%) exhibited enamel hypocalcification producing 124 defects, indicating a higher overall frequency of hypoplasia (Table 4.3). However, a standardizing defect ratio was determined by dividing the number of hypoplastic or hypocalcified defects by the total number of defective teeth. The result sees a more equal degree of defects (i.e. number of stress episodes) per defective teeth for hypoplasia (1.19) and hypocalcification

Table 4.3: Comparison of enamel defect frequency by permanent tooth type

	hypoplasia					hypocalcification				
	N	N_d	N_d/N	n	n/N_d	N	N_d	N_d/N	n	n/N_d
maxillary										
I1	9	6	0.67	6	1.00	9	7	0.78	13	1.86
I2	8	7	0.88	8	1.14	8	7	0.88	9	1.29
C	8	7	0.88	10	1.43	8	6	0.75	8	1.33
PM1	11	10	0.91	12	1.20	11	8	0.73	9	1.13
PM2	12	10	0.83	12	1.20	12	10	0.83	10	1.00
M1	10	8	0.80	8	1.00	10	8	0.80	8	1.00
M2	7	5	0.71	5	1.00	7	6	0.86	7	1.17
M3	6	6	1.00	8	1.33	6	5	0.83	8	1.60
mandibular										
I1	6	3	0.50	3	1.00	6	4	0.67	4	1.00
I2	8	6	0.75	7	1.17	8	4	0.50	4	1.00
C	11	10	0.91	12	1.20	11	8	0.73	10	1.25
PM1	12	11	0.85	13	1.18	13	8	0.62	8	1.00
PM2	13	10	0.77	11	1.10	13	7	0.54	7	1.00
M1	11	7	0.64	7	1.00	11	6	0.55	6	1.00
M2	11	7	0.64	11	1.57	11	6	0.55	7	1.17
M3	9	8	0.89	11	1.38	9	5	0.56	6	1.20
total	153	121	0.79	144	1.19	153	105	0.68	124	1.18

N = number of scorable teeth
N_d = number of teeth with one or more hypoplastic or hypocalcified defects
N_d/N = frequency of hypoplastic or hypocalcified teeth (number of defective teeth divided by the total number of teeth scored)
n = total number of hypoplastic or hypocalcified defects
n/N_d = defect ratio (number of defects divided by the total number of defective teeth)

(1.18). The maxillary and mandibular dentitions are relatively equally hypoplastic, but the maxillary teeth are more hypocalcified than are the mandibular.

Although the overall defect ratio points to a similar overall stress profile for hypoplasia and hypocalcification, the inter-tooth frequencies and defect ratios differ for each tooth type (Table 4.3). If considered by the frequency of defective teeth, the most hypoplastic tooth type is the maxillary third molar (100%), followed by the mandibular canine (91%) and the maxillary first premolar (91%). The least defective tooth type is the mandibular central incisor (50%). If considered by defect ratio (i.e. the number of defects divided by the number of defective teeth), then the mandibular second molar (1.57), followed by the

maxillary canine (1.43) and mandibular third molar (1.38) are the most hypoplastic. The least hypoplastic tooth types by number of defects are the maxillary and mandibular central incisors and first molars and the maxillary second molar, all having defect ratios of 1.00.

The most hypocalcified tooth types by frequency of defective teeth are the maxillary second incisor (88%) and second molar (86%) (Table 4.3). If considered by defect ratio, the maxillary central incisor (1.86) and third molar (1.60) are the most hypocalcified. The least hypocalcified tooth types are the mandibular lateral incisor (50%) and the second premolar (54%) by frequency of defective teeth, and by defect ratio the maxillary and mandibular second premolar and first molar and mandibular central and lateral incisors all having a defect ratio of (1.00).

Linear enamel hypoplasia (LEH) was the most prevalent type of hypoplastic defect (82%), followed by pitting (11%) and focal loss of enamel (7%) (Table 4.4). The majority of hypocalcified defects were of the diffuse type (95%).

Both classes of enamel defects were most often present on the labial/buccal surface (Table 4.5); 72% of the hypoplastic defects and 58% of the hypocalcified defects, although nearly half of the enamel opacities were found on the lingual surface (42%). Hypoplastic defects on the lingual surface tended to be more often in the maxillary and mandibular posterior dentition. The majority of hypoplastic defects involved the labial/buccal surface only (65%), followed by defects incorporating both surfaces (28%) and few isolated on the lingual surface (7%) (Table 4.6). In contrast, the majority of hypocalcified defects incorporated both the lingual and labial/buccal surfaces (45%), followed by defects restricted to the labial/buccal surface (40%).

Considering the labial surface only, hypoplastic defects are most often found in the cervical or middle thirds or involve both the cervical and middle thirds (Table 4.7). No hypoplastic defects involved both the middle and incisal/occlusal thirds. Similarly,

Table 4.4: Frequency and inter-tooth distribution by defect type

	hypoplasia							hypocalcification				
		pitting		LEH		focal loss			diffuse		demarcated	
	N_h	n	%	n	%	n	%	N_o	n	%	n	%
maxillary												
I1	6	0	-	6	(100)	0	-	13	13	(100)	0	-
I2	8	0	-	5	(63)	3	(37)	9	9	(100)	0	-
C	10	0	-	9	(90)	1	(10)	8	8	(100)	0	-
PMI	12	1	(8)	11	(92)	0	-	9	9	(100)	0	-
PM2	12	1	(8)	10	(84)	1	(8)	10	10	(100)	0	-
M1	8	1	(12)	6	(75)	1	(12)	8	8	(100)	0	-
M2	5	0	-	4	(80)	1	(20)	7	6	(86)	1	(14)
M3	8	2	(25)	6	(75)	0	-	8	6	(75)	2	(25)
mandibular												
I1	3	0	-	3	(100)	0	-	4	4	(100)	0	-
I2	7	1	(14)	5	(71)	1	(14)	4	4	(100)	0	-
C	12	0	-	10	(83)	2	(17)	10	9	(90)	1	(10)
PM1	13	2	(15)	11	(85)	0	-	8	8	(100)	0	-
PM2	11	1	(9)	10	(91)	0	-	7	7	(100)	0	-
M1	7	0	-	7	(100)	0	-	6	6	(100)	0	-
M2	11	3	(27)	8	(73)	0	-	7	6	(86)	1	(14)
M3	11	4	(36)	7	(64)	0	-	6	5	(83)	1	(17)
total	144	16	(11)	118	(82)	10	(7)	124	118	(95)	6	(5)

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects per tooth third or tooth third combination
% = frequency of hypoplastic or hypocalcified defects per tooth third or tooth third combination

A comparison of the frequency and intertooth distribution of hypoplastic and hypocalcified defect types, considering both lingual and labial surface defects.

hypocalcified defects were most prevalent in the cervical or middle thirds, or involved both the cervical and middle thirds (Table 4.8). The maxillary anterior teeth have a higher frequency of hypocalcification in the incisal third, as well as incorporating the entire labial surface.

Table 4.5: Defect frequency and inter-tooth distribution by surface

	hypoplasia			hypocalcification		
		lingual	labial		lingual	labial
	N_h	n (%)	n (%)	N_o	n (%)	n (%)
maxillary						
I1	6	0 -	6 (100)	15	6 (40)	9 (60)
I2	9	1 (11)	8 (89)	15	6 (40)	9 (60)
C	11	3 (11)	8 (73)	13	6 (46)	7 (54)
PMI	15	4 (27)	11 (73)	11	6 (55)	5 (45)
PM2	17	6 (35)	11 (65)	16	8 (50)	8 (50)
M1	10	2 (20)	8 (80)	12	4 (33)	8 (67)
M2	7	2 (29)	5 (71)	9	4 (44)	5 (56)
M3	9	3 (33)	6 (67)	9	4 (44)	5 (56)
mandibular						
I1	3	0 -	3 (100)	6	2 (33)	4 (67)
I2	7	1 (14)	6 (86)	5	1 (20)	4 (80)
C	17	5 (29)	12 (71)	16	6 (37)	10 (63)
PM1	19	7 (37)	12 (63)	14	6 (43)	8 (57)
PM2	18	8 (44)	10 (56)	13	7 (54)	6 (46)
M1	8	1 (12)	7 (88)	9	3 (33)	6 (67)
M2	13	4 (31)	9 (69)	10	4 (40)	6 (60)
M3	16	5 (31)	11 (69)	7	2 (29)	5 (71)
total	185	52 (28)	133 (72)	180	75 (42)	105 (58)

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects present per surface
% = frequency of hypoplastic or hypocalcified defects present per surface

A comparison of hypoplastic and hypocalcified defect frequency and inter-tooth distribution by surface presence. Note defects incorporating both the lingual and labial surfaces will be counted as one lingual and one labial defect.

Discussion

Defect frequency and distribution in the Tell Leilan permanent sample reveals data consistent with known intra-tooth variability in defect expression but conflicts with inter-tooth findings from previous studies (Black 1979; Cutress and Suckling 1982; El-Najjar et al. 1978; Goodman and Armelagos 1985b; Moggi-Cecchi et al. 1994; Needleman et al. 1991; Smith and Peretz 1986). Intra- and inter-tooth variability in defect frequency and

Table 4.6: Defect frequency and distribution by surface incorporation

	hypoplasia							hypocalcification						
		lingual		labial		both			lingual		labial		both	
	N_h	n	(%)	n	(%)	n	(%)	N_o	n	(%)	n	(%)	n	(%)
maxillary														
I1	6	0	-	6	(100)	0	-	13	4	(31)	7	(54)	2	(15)
I2	8	0	-	7	(87)	1	(13)	9	0	-	3	(33)	6	(67)
C	10	2	(20)	7	(70)	1	(10)	8	1	(13)	2	(25)	5	(62)
PMI	12	1	(8)	8	(67)	3	(25)	9	4	(45)	3	(33)	2	(22)
PM2	12	1	(8)	6	(50)	5	(42)	10	2	(20)	2	(20)	6	(60)
M1	8	0	-	6	(75)	2	(25)	8	0	-	4	(50)	4	(50)
M2	5	0	-	3	(60)	2	(40)	7	2	(29)	3	(42)	2	(29)
M3	8	2	(25)	5	(62)	1	(13)	8	3	(37)	4	(50)	1	(13)
mandibular														
I1	3	0	-	3	(100)	0	-	4	0	-	2	(50)	2	(50)
I2	7	1	(14)	6	(86)	0	-	4	0	-	3	(75)	1	(25)
C	12	0	-	7	(58)	5	(42)	10	0	-	4	(40)	6	(60)
PM1	13	1	(8)	6	(46)	6	(46)	8	0	-	2	(25)	6	(75)
PM2	11	1	(10)	3	(30)	7	(70)	7	1	(14)	0	-	6	(86)
M1	7	0	-	6	(86)	1	(14)	6	0	-	3	(50)	3	(50)
M2	11	2	(18)	7	(64)	2	(18)	7	1	(14)	3	(43)	3	(43)
M3	11	0	-	6	(54)	5	(46)	6	1	(17)	4	(66)	1	(17)
total	144	10	(7)	93	(65)	41	(28)	124	19	(15)	49	(40)	56	(45)

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects involving the surface(s)
% = frequency of hypoplastic or hypocalcified defects involving the surface(s)

A comparison of the frequency and inter-tooth distribution of hypoplastic and hypocalcified defects involving the lingual, labial/buccal or both surfaces.

distribution are considered to reflect relative sensitivity or susceptibility of different tooth types and tooth areas to physiological insult. In a review of past studies discussing dissimilarity in hypoplastic defect expression, Goodman and Armelagos (1985b:480) note that the variation in defect frequency between tooth types is "...common and substantial...", and provide an inter-tooth analysis of hypoplastic defect frequency and distribution. Testing the assumption of "uniformity of response" of different tooth types (i.e. developing tooth crowns will be equally exposed and responsive to stress), their findings suggest that defect frequency varies substantially between tooth types as well as within individual tooth crowns.

Table 4.7: Hypoplastic defect frequency by tooth third location

		cervical		middle		incisal		cervical & middle		middle & incisal		entire tooth crown	
	N	n	%	n	%	n	%	n	%	n	%	n	%
maxillary													
I1	6	0	-	2	(33)	0	-	4	(67)	0	-	0	-
I2	8	1	(12)	4	(50)	1	(12)	2	(25)	0	-	0	-
C	8	2	(25)	1	(12)	0	-	5	(63)	0	-	0	-
PMI	11	3	(27)	7	(64)	1	(9)	0	-	0	-	0	-
PM2	11	4	(36)	4	(36)	1	(10)	2	(18)	0	-	0	-
M1	8	4	(50)	4	(50)	0	-	0	-	0	-	0	-
M2	5	3	(60)	1	(20)	0	-	1	(20)	0	-	0	-
M3	6	5	(83)	0	-	0	-	1	(17)	0	-	0	-
mandibular													
I1	3	1	(33)	2	(67)	0	-	0	-	0	-	0	-
I2	7	1	(14)	4	(57)	0	-	2	(29)	0	-	0	-
C	12	2	(17)	4	(33)	0	-	5	(42)	0	-	1	(8)
PM1	12	6	(50)	5	(42)	0	-	0	-	0	-	1	(8)
PM2	10	3	(30)	5	(50)	0	-	2	(20)	0	-	0	-
M1	8	5	(63)	1	(12)	0	-	1	(12)	0	-	1	(12)
M2	9	3	(33)	5	(56)	0	-	1	(11)	0	-	0	-
M3	11	4	(36)	2	(17)	4	(36)	1	(9)	0	-	0	-
total	135	47	(35)	51	(38)	7	(5)	27	(20)	0	-	3	(2)

N = total number of hypoplastic defects per tooth type
n = number of hypoplastic defects per tooth third or combination of tooth thirds
% = frequency of hypoplastic defects per tooth third or combination of tooth thirds

Table showing the frequency and intertooth distribution by tooth third of hypoplastic defects present on the labial surface.

Several theories have been proposed to explain intra- and inter-tooth variation in hypoplastic, as well as hypocalcified, defect expression: a hypoplastic defect is formed only when the magnitude of stress for a given tooth type reaches a critical threshold (Goodman and Rose 1990); variation in the rate of enamel deposition between and within tooth types contributes to variation in defect expression (Condon and Rose 1992; Fearne et al. 1994; Needleman et al. 1991; Suga 1989); and crown geometry, namely appositional versus imbricational zones of crown growth and spacing between developmental layers, is related to hypoplastic expression (Hillson and Bond 1997; Guatelli-Steinberg and Lukacs 1998).

Table 4.8: Hypocalcified defect frequency by tooth third location

		cervical		middle		incisal		cervical & middle		middle & incisal		entire tooth crown	
	N	n	%	n	%	n	%	n	%	n	%	n	%
maxillary													
I1	9	2	(22)	0	-	2	(22)	2	(22)	1	(12)	2	(22)
I2	9	4	(44)	0	-	2	(22)	2	(22)	0	-	1	(12)
C	7	2	(29)	0	-	1	(13)	2	(29)	0	-	2	(29)
PMI	5	1	(20)	1	(20)	0	-	2	(40)	0	-	1	(20)
PM2	8	3	(38)	1	(12)	0	-	4	(50)	0	-	0	-
M1	8	6	(75)	1	(12)	0	-	1	(12)	0	-	0	-
M2	5	4	(80)	0	-	0	-	1	(20)	0	-	0	-
M3	5	3	(60)	1	(20)	0	-	1	(20)	0	-	0	-
mandibular													
I1	4	3	(75)	1	(25)	0	-	0	-	0	-	0	-
I2	4	3	(75)	1	(25)	0	-	0	-	0	-	0	-
C	10	2	(20)	1	(10)	1	(10)	5	(50)	0	-	1	(10)
PM1	8	4	(50)	1	(12)	0	-	2	(25)	1	(12)	0	-
PM2	6	4	(67)	2	(33)	0	-	0	-	0	-	0	-
M1	6	5	(83)	1	(17)	0	-	0	-	0	-	0	-
M2	6	5	(83)	0	-	0	-	1	(17)	0	-	0	-
M3	5	2	(40)	2	(40)	0	-	1	(20)	0	-	0	-
total	105	53	(50)	13	(12)	6	(6)	24	(23)	2	(2)	7	(7)

N = total number of hypocalcified defects per tooth type
n = number of hypocalcified defects per tooth third or tooth third combination
% = frequency of hypocalcified defects per tooth third or tooth third combination

Table showing the frequency and intertooth distribution by tooth third of hypocalcified defects present on the labial surface.

Intra-tooth variation in the Tell Leilan sample reveals a higher prevalence of hypoplastic and hypocalcified defects on the labial/buccal surface and in the cervical and middle thirds of each tooth type. Although this intra-tooth distribution is in agreement with previous studies (e.g. Condon and Rose 1992; Goodman and Armelagos 1985b; Needleman et al. 1991; Smith and Peretz 1986), defect frequency by permanent tooth type is inconsistent. Most studies determine the most frequently hypoplastic or hypocalcified tooth type by the number of teeth with at least one defect, divided by the number of scorable teeth. These studies have found the anterior teeth, especially the maxillary, to be more hypoplastic and hypocalcified than the posterior teeth (Al-Abbasi and Sarie' 1997; Cucina et al. 1996;

Goodman and Armelagos 1985b; Needleman et al. 1991; Smith and Peretz 1986; Wright 1997). This is not the case for hypoplasia in the Tell Leilan sample. Neither the maxillary nor mandibular dentitions exhibit differences in frequency of hypoplasia between the anterior and posterior teeth. Furthermore, studies have found the maxillary central incisor and mandibular canine to be the most hypoplastic tooth types (e.g. Al-Abbasi and Sarie' 1997; Cucina et al. 1996; Goodman and Armelagos 1985b). In the Tell Leilan sample, although the mandibular canine is the most hypoplastic of the mandibular dentition, the central incisor is the least defective and the third molar the most hypoplastic in the maxillary dentition. Many studies do not include the maxillary and mandibular third molars (e.g. Goodman and Armelagos 1985b; Wright 1997), however the few studies that do have not found higher frequencies of hypoplasia relative to the anterior teeth (Al-Abbasi and Sarie' 1997; Cucina et al. 1996). If the third molars are excluded from the Tell Leilan sample, then the first premolar is the most frequently hypoplastic maxillary tooth type.

If inter-tooth susceptibility is considered by the number of hypoplastic defects per defective tooth, then the maxillary canine and the mandibular second molar exhibit the highest number of stress episodes. Again, this contradicts previous findings. Goodman and Armelagos (1985b) calculated the number of hypoplastic defects per tooth type and found the anterior maxillary teeth, namely the central incisor, to be the most affected. In the Tell Leilan dental sample, the lower frequency of hypoplasia for the maxillary central incisor, as well as for anterior versus posterior teeth, is likely due to the small sample size, and the fact that individuals are represented by incomplete dentitions.

In contrast to hypoplastic defects in this study, enamel opacities are most frequently found on the labial surface of the maxillary teeth, with the lateral incisor being the most frequently hypocalcified tooth type. This frequency and distribution is consistent with epidemiological data on hypocalcified defect expression (Forrest and James 1965; Murray and Shaw 1979; Nevitt et al. 1963). However, the maxillary posterior teeth are more hypocalcified than are the anterior, which is opposite to the distribution of opacities found

by Needleman et al. (1991). If an inter-tooth comparison is based on the number of hypocalcified defects per defective tooth type (i.e. defect ratio), then more defects are found in the maxillary anterior teeth as compared to the posterior, and the maxillary central incisor exhibits the most hypocalcified defects.

In contrast to the conclusions from the analysis of hypoplasia, hypocalcification data suggest a different pattern of stress; namely, different inter-tooth frequencies of defective teeth and different numbers of stress episodes. Various factors may explain these conflicting results. First, in a discussion of the limited number of anthropological studies analyzing hypocalcification as an indicator of non-specific stress, Hillson (1996) notes that the concern of potential post-mortem/taphonomic influences, which may affect enamel color or translucency, may deter their use. Enamel hypoplasia has long been considered significantly more resistant to alteration in the burial environment; Rose et al. (1985:285) discuss that as early as Tomes (1892, cited in Rose et al. 1985), studies have found enamel "...virtually unaltered..." in the burial environment and, subsequently, hypoplastic defects were considered the result of metabolic disturbances. Post-mortem conditions in the burial environment may involve leaching of the mineral content of teeth (e.g. root etching). Since enamel hypocalcification results in less mineralized enamel with a higher organic content (Crenshaw and Bawden 1984; Suga 1989), distinguishing between post-mortem and ante-mortem causes of lower mineralization may be of concern. Furthermore, as Needleman et al. (1991:213) note, "...prolonged desiccation..." may make opacities easier to detect or even artificial. This statement is supported by other findings: in discussing the location of diffuse opacities, Suckling et al. (1989:226) state that most defects were found most often on the "...cusps and occlusal ridges but were more widely distributed over the rest of the crown when the enamel was dry...".

Second, antemortem conditions can also affect the frequency and distribution of enamel hypocalcification. Needleman et al. (1991:213) note the potential of dental calculus to decalcify enamel and produce "white spot" decalcification which can imitate enamel hypocalcification. As Croll (1991:22, 26) explains, enamel hypocalcification is

developmental since it is the result of a disturbance during the mineralization phase of enamel development. Enamel decalcification, on the other hand, is *acquired* and occurs when "...dental plaque persists undisturbed on an enamel surface and the organic acids that are subsequently produced etch the mineral content out of the enamel surface...". Acquired enamel decalcification is usually seen in the cervical region of all teeth (i.e. common sites for dental calculus) (Croll 1991) and for this reason, Needleman et al. (1991) excluded opaque defects found in this region from their analyses, presumably since there would be no way of differentiating between developmental and acquired defects. Initial preparation of the Tell Leilan dental sample did include scoring and removal of dental calculus. If dental calculus did indeed influence hypocalcification frequency and distribution, then this would explain the higher frequency of defects present on the lingual surface, as well as defects involving both the lingual and labial/buccal surfaces since calculus is often found on both surfaces. Furthermore, Needleman et al. (1991:215) consider the high frequency of enamel opacities on the labial surface of the maxillary anterior teeth may also be the result of "mechanical trauma" to developing teeth. These authors propose that due to the position in the oral cavity and within the dental arch, the middle third of the labial surface of developing maxillary anterior teeth "...may be more susceptible to mechanical insults..." through the thin cortical plate of the maxilla.

Due to contrasting evidence from the analysis of enamel hypocalcification as compared to hypoplasia, as well as in light of possible non-developmental factors acting on prevalence and distribution, the use of enamel hypocalcification as an indicator of non-specific stress should be reconsidered.

Conclusion

A comparative analysis of hypoplastic and hypocalcified defect frequency and distribution in the Tell Leilan dental sample suggest different patterns of non-specific stress. Although intra-tooth variation is consistent with previous studies, inter-tooth defect patterns differ between hypoplasia and hypocalcification. Two factors may be influencing this differential

patterning: postmortem alteration can creating pseudo-opacities; and antemortem conditions, such as dental calculus or localized trauma, producing opacities that are *acquired* rather than *developmental*. With the potential effects of non-developmental factors influencing hypocalcified defect expression, the use of enamel hypocalcification as an indicator of non-specific stress should be reconsidered.

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Chapter 5:
The health impact of climate change at Tell Leilan (Syria): a multi-level analysis of enamel hypoplasia

Introduction

Clinical confirmation of the link between enamel hypoplasia and non-specific stress (Kreshover 1960), combined with the epidemiological application of assessing demographic and socioeconomic patterns of disease (e.g. Cutress and Suckling 1982; Goodman et al. 1987; May et al. 1993), have made the analysis of hypoplasia popular in anthropology. Furthermore, the durability of dental enamel in the burial environment and its permanent recording of a physiological insult in the form of an enamel defect have made dental indicators of stress more reliable than skeletal markers for assessing health and stress profiles (Goodman and Armelagos 1985a; Goodman et al. 1984; Hush-Ashmore et al. 1982; Rose et al. 1985). Consequently, the analysis of developmental defects of enamel as indicators of non-specific stress has allowed anthropologists to construct stress profiles for archaeological populations and to investigate the effect of cultural change on health. Various themes of such an investigation include: the transition from a hunter-gatherer subsistence to agriculture (see Cohen and Armelagos 1984), the health and stress associated with American slavery (Blakely et al. 1994), the arrival of Europeans in the New World (Hutchinson and Larsen 1990; Santos and Coimbra 1999; Ubelaker 1994), and the rise of industrialization and urbanization during the 18th and 19th centuries (Wood 1996).

Another area of investigation is the examination of historical and archaeological models of culture change (e.g. Wright 1997). Researchers often debate the cause of the collapse of past civilizations and, subsequently, several explanatory models are proposed. One such debate is the cause of the collapse of the Akkadian occupation in northern Mesopotamia at

the end of the third millennium BC. The societal collapse is evident with the abandonment of numerous sites including the multi-period site of Tell Leilan in northeastern Syria (Figure 5.1).

Following a long and dynamic culture history, the site of Tell Leilan was abandoned for a period of 300 years (~2200-1900 BC) (Weiss et al. 1993). The period of abandonment, referred to as the "Habur hiatus 1", is considered by Weiss et al. (1993:999) to be the result of climate change severe enough to disrupt the traditional dry-farming agricultural base. Paleoclimatological evidence from Tell Leilan does indicate a period of marked aridity preceding the abandonment of the site and lasting until reoccupation during the late second millennium. Weiss et al. (1993:1002) point out that climate change and Akkadian collapse are "...synchronous with climate change and collapse phenomena documented in the Aegean, Egypt, Palestine and the Indus..." (e.g. Esse 1989; Rosen 1989, 1995) and that historical records (i.e. clay tablets) chronicle a migration of northern peoples into southern Mesopotamia as evident in an influx of northern tribal names in southern cities (Gibbons 1993). To further investigate the claim of Weiss et al. (1993), this chapter will assess the possible health impact of climate change at Tell Leilan through a multi-level analysis of enamel hypoplasia.

The potential effects of climate change on stress patterns

Six general periods reflecting cultural development and change can be identified at Tell Leilan through material culture analysis; Hassuna/Halaf, Ubaid, Uruk, Ninevite, the Akkadian occupation, and the reign of Shamshi-Adad. In reviewing the political and socioeconomic aspects of each cultural period, three possible themes of change can be identified with the transition from one period to the next: a shift in population size and density; a transformation of social organization and stratification; and an alteration of climatic conditions (Table 5.1).

All three of these changes could directly affect the stress profile for a population. A shift in population size and/or density would affect the likelihood of infectious disease

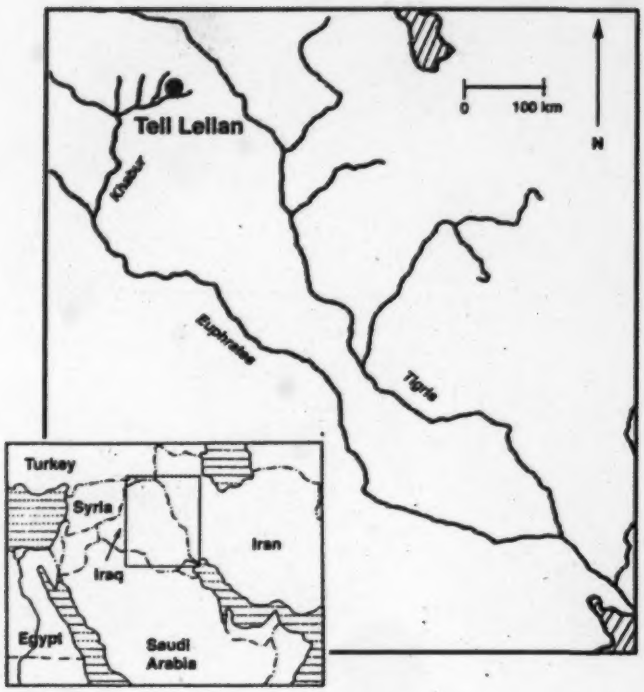

Figure 5.1: Regional map of Mesopotamia identifying Tell Leilan (map adapted from Stein 1994:13; inset from Bernbeck 1995:10).

transmission (e.g. Kent and Dunn 1996), and, due to its synergistic relationship with malnutrition, hamper adequate nutrition. Increased social stratification could target stress on certain segments of the society (e.g. infectious disease in crowded lower class conditions) and within a specific group (e.g. infectious disease among the lower classes that exacerbated malnutrition). Conversely, a decrease in social stratification would distribute the stress profile more evenly across the society with individuals having more equal access to resources and more similar living conditions. A change in climatic conditions could affect the agricultural base directly, thereby disrupting dietary adequacy, and/or encouraging or discouraging certain infectious diseases.

Table 5.1: Chronology of Tell Leilan including general periods, Leilan periods, radiocarbon dates and possible influences on health and stress (based on Bahn 1992; Weiss 1990; Weiss et al. 1993)

General period	Leilan period	Date (BC)	Factors affecting health
Reign of Shamshi-Adad	I	~1990-1728	lower density city, dense distribution of village settlements
Habur hiatus 1	-----	~2200-1900	abandonment; desertification
Akkadian imperialism	IIb	~2300-2200	densely inhabited city; socially stratified; agricultural intensification; climate change
Urban period	IIa	~2400-2300	further increase in social stratification
Ninevite V			
late	IIId	~2600-2400	increase in site size; increased social stratification
middle	IIIb-c	~2900-2600	increase in social stratification?
early	IIIa	~3000-2900	dispersed low density settlements; decreased social stratification
Uruk			
late	IV	~3300-3000	increase in settlement size; increased social stratification
early	V	~4100-3300	
Ubaid (Chalcolithic)	VI	~5500-4100	increased sedentarism and population densities; towns coalesced along rivers
Halaf	-----	~late 6th-early 5th millennium	small settlement size; some increase in social stratification (chiefdoms?)
Hassuna (Neolithic)	-----	~6th millennium	small village-level settlements; minimal social stratification

The direct effects of climate change, such as famine, would be evident in a stress profile of a population (e.g. Zhou and Corruccini 1997). However, secondary effects of climate change would be indistinguishable from the direct effects and could have also increased the pathogen load in several ways. In the case of famine, for example, a population migration from the rural community to the city in search of food could increase the population density of the lower classes facilitating increased disease transmission and also

increasing the stress on food reserves. If the social response to the climatic change is inadequate or maladaptive, then a series of secondary effects could ultimately lead to the collapse of the society and the abandonment of the site (see Rosen 1995). However, since a stress profile is based on indicators of *non-specific* stress, how can the stress resulting from climate change be distinguished from other forms of stress (i.e. changes in population size and density, or social stratification)?

Cultural changes such as an increase or decrease in population size and density would affect the *frequency of stress*, as reflected in the frequency of enamel defects, and changes in social stratification could target specific segments of the society thereby altering the *distribution of stress* within the society. Climate change would have directly affected the dry-farming agricultural base of Tell Leilan and, therefore, affected all segments of the society, albeit possibly to varying degrees. With all or nearly all of the population being affected by the climatic change, with its direct and secondary effects, one would expect to see not only an increase in the frequency of affected individuals but also in a broader distribution among the population for the period preceding the abandonment of the site (period II) as compared to previous periods (periods III and VI) and the subsequent reoccupation of the site (period I).

Materials and Methods

The Tell Leilan dental sample consists of 183 permanent teeth, both maxillary and mandibular. Since the methodology chosen for this study involves a multi-level analysis of the entire tooth crown, the selection of dental specimens to be analyzed was based on two factors. First, teeth with more than slight wear (stage 2 of Smith's methodology, Smith 1984:45) were excluded since moderate to severe attrition would obliterate any evidence of defects in the occlusal third of the crown. Furthermore, teeth with antemortem conditions such as caries or chipping were also deleted from the sample. Second, teeth revealing postmortem damage such as chipping, cracking or enamel loss within one of the three areas was also deleted from the sample.

In order to optimize the number of scorable teeth, dental calculus was removed after being scored according to the method described by Buikstra and Ubelaker (1994:56). A wooden probe was used for removal in order to minimize damage (e.g. scraping or grooving) to the enamel surface. All teeth were further cleaned by dry brushing to remove remnant calculus and dirt. In cases where the dental and skeletal remains had been consolidated in the field (e.g. with polyvinyl acetate), acetone applied by a cotton swab or brush was used to remove the consolidant. This selection and cleaning process yielded a final dental sample of 153 permanent teeth representing 22 individuals.

Due to the limited number of individuals for each archaeological period (Table 5.2), period II (i.e. the period preceding abandonment of the site) will be compared to periods I, III and VI combined. Although each archaeological period would have specific cultural aspects influencing stress, these influences fall into the three general categories; changes in social stratification, changes in population size and density, and climatic change. All periods share changes in social organization and settlement size and density but only period II has the effects of climate change. Since it is the focus of this study to investigate the health impact of climate change, any notable differences in stress frequencies between period II and the other periods will be evaluated as possibly climatic in nature.

Since the dental specimens were recovered from intramural burial contexts, care was taken to ensure that all teeth belonged to the same individual and that no teeth belonging to the same individual were counted as two individuals. Sex was indeterminate for most individuals and, therefore, not considered in this study, and only general age categories (i.e. child, adolescent, adult) could be assigned. Furthermore, individual status was also not evident (i.e. no distinguishing burial artifacts), thereby preventing any consideration of health and status.

Table 5.2: Number of individuals and scorable teeth by archaeological period

	period					
	I	II	III	VI	I, III &VI	total
total number of individuals	1	**15**	5	1	7	22
total number of scorable teeth	5	**101**	42	5	52	153

Table showing the number of individuals and scorable teeth for each archaeological period. Note the disparity between the sample sizes for period II (in bold) and the other periods.

Hypoplastic defects were assessed visually on all available permanent teeth. Although a closer examination of defective enamel included the use of a light microscope and a hand lens, only defects seen by the naked eye were recorded. Whereas several researchers (e.g. Buikstra and Ubelaker 1994) suggest analysis of one or two commonly defective tooth types (e.g. maxillary central incisor and mandibular canine) or the frequently defective anterior dentition, this study analyzed all tooth types independently to provide a larger dental sample and a greater age range (i.e. prenatal to ~15 years for crown formation). By analyzing each tooth type independently, inter-tooth variability in defect expression may be accounted for. The left antimere of each tooth type was chosen for analysis. When the left antimere was not scorable, then the right antimere was used.

The methodology for this study is based on the standard method provided by Buikstra and Ubelaker (1994:56-58) for anthropological analyses of archaeological remains, and the Developmental Defects of Enamel (DDE) index proposed by the Federation Dentaire Internationale (1982) for use in epidemiological studies of enamel defects. As with both methods, defect type and location were recorded. However, the location of a defect was noted in a way different to both methods. In the method provided by Buikstra and Ubelaker (1994), "location" refers to measuring the position of the defect from the

cemento-enamel junction (CEJ) to the most occlusal limit of the defect. This measurement is subsequently converted into an "age of occurrence" through standardized tables. Due to concern about intra- and inter-tooth variation in defect frequency and expression (Cuttress and Suckling 1982; El-Najjar et al. 1978; Goodman and Armelagos 1985b; Hillson and Bond 1997), and since the primary aim of this analysis is to investigate stress frequency and distribution among individuals and within the society, rather than age-specific health patterns, this study did not calculate a mean "age of occurrence" to determine a peak age of stress. In the DDE index, "location" is defined by tooth areas; gingival half, incisal half, occlusal, cuspal or the whole crown surface. The method used in this study redefined tooth area as cervical, middle and occlusal thirds, to be compatible with the study by Goodman and Armelagos (1985b).

Although "age of occurrence" was not calculated, age-at-death categories (i.e. child, adolescent, adult) were tabulated in order that a relationship between systemic stress (indicated by enamel hypoplasia) and mortality could be investigated.

Hypoplastic defects were recorded as to type, surface and location (Table 5.3). A defect is defined as any irregularity or abnormality in the dental enamel. Though the thickness varies from cusp to tip, the surface is smooth overall with the only ridging (i.e. perikymata) occurring naturally and at the microscopic level. Any alteration in the quantity of dental enamel, due to a metabolic or physiological insult, results in a developmental enamel defect such as hypoplasia.

Enamel hypoplasia is defined as any reduction in the quantity of dental enamel and is the result of a systemic disruption during the secretory phase of enamel development. It can appear as a single pit or as a linear array of pits which are recorded collectively under "pits". Horizontal grooves, also known as "linear enamel hypoplasia" (LEH) (Figure 5.2), are also indicative of enamel hypoplasia and may appear on one surface (the lingual or labial/buccal surfaces) or circumscribe the tooth (both surfaces). A focal loss of enamel is defined as an area of enamel reduction that is not a pit or a groove. The etiological and

Table 5.3: Dental enamel defects data collection key (based on Buikstra and Ubelaker 1994; FDI 1982)

A. defect	- = not present 0 = unscorable 1 = normal enamel 2 = hypoplastic enamel
B. type	1 = pitting 2 = horizontal grooves (LEH) 3 = focal loss of enamel
C. surface	1 = lingual 2 = labial/buccal 3 = both surfaces
D. location	1 = cervical third 2 = middle third 3 = incisal/occlusal third 4 = cervical and middle thirds 5 = middle and incisal thirds 6 = entire tooth crown

microstructural differences between hypoplastic lesions are not fully understood (for a recent discussion and proposed explanation, see Hillson and Bond 1997).

Defects were recorded as to their presence on the lingual or labial/buccal surface and as to surface incorporation (involving the lingual or labial/buccal surface only, or both surfaces). Due to interproximal wear and lack of full view for teeth still in situ in the maxilla or mandible, the mesial and distal surfaces were not considered. The locations of hypoplastic defects are further based on a division of the tooth crown into three sections equal in height: cervical, middle and incisal/occlusal. If a defect occupies more than one tooth third, it is recorded for all areas it incorporates (e.g. cervical and middle, middle and incisal/occlusal, or the entire surface). For comparability with other studies and since defects are most commonly found on the labial surface, defect location was scored on the labial surface only.

Figure 5.2: Linear enamel hypoplasia (LEH) on the labial surface of a mandibular incisor.

In order to address the hypothesis – that the health impact of climate change would be evident not only in an increase in the frequency of stress but in its distribution across the population as well - data gained from this study was analyzed by individual and by tooth type. At the level of the individual (i.e. individual count), any changes in the distribution or pattern of hypoplasia within the population can be identified and compared between the periods, as well as within age specific categories. By comparing defect prevalence between tooth types (i.e. tooth count), changes in defect frequency may be ascertained.

This method provides a multi-level analysis of enamel hypoplasia. It is anticipated that by investigating defect frequency and distribution at both the level of the individual and by

tooth-type not only will a change in the frequency and distribution of stress be determined, but any associated variations in the pattern of defect expression may also be identified.

Results

The distribution of stress across the population was determined by the number of individuals (i.e. individual count) with one or more hypoplastic defects. Overall, the prevalence of hypoplasia in period II (87%) is less than that for periods I, III and VI combined (100%). However, a comparison of the prevalence of hypoplasia for different age-at-death categories (i.e. child, adolescent and adult) indicates that there is no difference in prevalence among adults (Figure 5.3, Table 5.4). Unfortunately, since there are no subadults in periods I, III or VI, any differences in the frequency of stress among children and adolescents are unknown.

An inter-tooth analysis (i.e. tooth count) reveals no differences in hypoplastic defect frequency between the periods (Table 5.5). The overall frequency of teeth with at least one hypoplastic defect is the same for both period II and periods I, III and VI combined (79%). A comparison of the frequency of hypoplasia by tooth type, however, reveals an increase in the frequency of defective teeth for certain tooth types but a decrease for other tooth types for period II.

In order to compare the number of stress episodes experienced in each of the time periods, a standardizing defect ratio was determined for each tooth type by dividing the number of hypoplastic defects by the number of defective teeth. Period II reveals a higher overall defect ratio (1.21) as compared to the other periods (1.15) indicating a greater number of hypoplastic defects per defective tooth (Table 5.5). An inter-period comparison of defect ratios by tooth type reveals a greater defect ratio for four tooth types in period II. However, periods I, III and VI combined exhibit higher hypoplastic defect ratios for three tooth types: the maxillary and mandibular third molars and the mandibular second molar. There is no difference in defect ratios for five tooth types.

Table 5.4: Frequency of hypoplasia by age group

	period					
	II			I, III & VI		
	N	N_h	%	N	N_h	%
child	3	1	33	0	-	-
adolescent	3	3	100	0	-	-
adult	8	8	100	6	6	100
unknown	1	1	100	1	1	100
total	15	13	87	7	7	100

N = the total number of individuals for each age-at-death category
N_h = number of individuals with at least one hypoplastic defect
% = the frequency of individuals with at least one hypoplastic defect for each age-at-death category

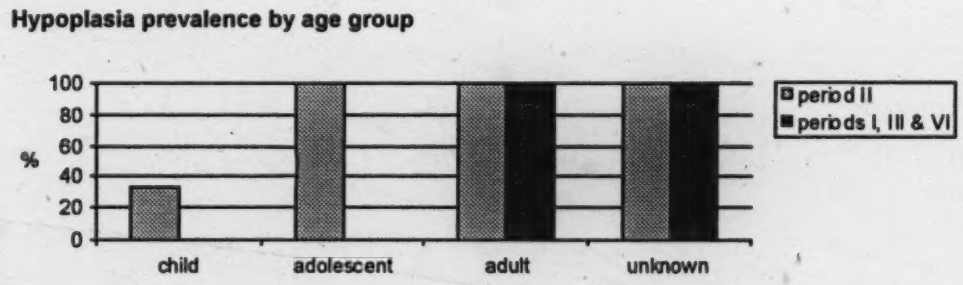

Figure 5.3: An inter-period comparison of the frequency of individuals with at least one hypoplastic defect. Note the equally high prevalence of hypoplasia for all periods in the adult group.

An inter-period comparison of hypoplastic defect type reveals LEH as the prominent form of defect expression in both period II (79%) and periods I, III and VI combined (88%) (Table 5.6). Period II reveals a higher frequency of pitting (13%) as compared to the other periods (6%), which is primarily exhibited in more posterior tooth types. The frequency of focal loss of enamel is similar

Table 5.5: Inter-period comparison of hypoplastic defect prevalence

	II					I, III &VI				
	N	N_d	N_d/N	n	n/N_d	N	N_d	N_d/N	n	n/N_d
maxillary										
I1	6	5	0.83	5	1.00	3	1	0.33	1	1.00
I2	4	4	1.00	5	1.25	4	3	0.75	3	1.00
C	5	4	0.80	6	1.50	3	3	1.00	4	1.33
PMI	5	5	1.00	7	1.40	6	5	0.83	5	1.00
PM2	7	6	0.86	8	1.33	5	4	0.80	4	1.00
M1	8	6	0.75	6	1.00	2	2	1.00	2	1.00
M2	5	4	0.80	4	1.00	2	1	0.50	1	1.00
M3	5	5	1.00	6	1.20	1	1	1.00	2	2.00
mandibular										
I1	4	2	0.50	2	1.00	2	1	0.50	1	1.00
I2	7	5	0.71	6	1.20	1	1	1.00	1	1.00
C	7	6	0.86	8	1.33	4	4	1.00	4	1.00
PM1	8	7	0.88	9	1.29	5	4	0.80	4	1.00
PM2	8	6	0.75	7	1.17	5	4	0.80	4	1.00
M1	10	6	0.60	6	1.00	1	1	1.00	1	1.00
M2	8	5	0.63	7	1.40	3	2	0.67	4	2.00
M3	4	4	1.00	5	1.25	5	4	0.80	6	1.50
total	101	80	0.79	97	1.21	52	41	0.79	47	1.15

N = number of scorable teeth
N_d = number of teeth with one or more hypoplastic defects
N_d/N = frequency of hypoplastic teeth (number of defective teeth divided by the total number of teeth scored)
n = total number of hypoplastic defects
n/N_d = defect ratio (number of defects divided by the total number of defective teeth)

for both period II (7%) and the combined periods I, III and VI (6%), and is found in both the anterior and posterior teeth.

The distribution of hypoplastic defects by surface is relatively the same for all periods (Table 5.7). In period II, 28% of the defects are present on the lingual surface and 72% on the labial/buccal. In periods I, III and VI combined, 27% are present on the lingual surface and 73% on the labial/buccal. However, if the frequency of surface presence is considered by tooth type, period II has a greater number of tooth types with lingual defects than the other periods, especially in the maxillary posterior teeth.

Table 5.6: Inter-period comparison of defect type

	II						I, III & VI					
		pitting		LEH		focal loss		pitting		LEH		focal loss
	N	n	(%)	n	(%)	n (%)	N	n	(%)	n	(%)	n (%)
maxillary												
I1	5	0	-	5	(100)	0 -	1	0	-	1	(100)	0 -
I2	5	0	-	3	(60)	2 (40)	3	0	-	2	(67)	1 (33)
C	6	0	-	5	(83)	1 (17)	4	0	-	4	(100)	0 -
PMI	7	1	(14)	6	(86)	0 -	5	0	-	5	(100)	0 -
PM2	8	1	(12)	6	(76)	1 (12)	4	0	-	4	(100)	0 -
M1	6	1	(17)	5	(83)	0 -	2	0	-	1	(50)	1 (50)
M2	4	0	-	4	(100)	0 -	1	0	-	0	-	1 (100)
M3	6	2	(33)	4	(67)	0 -	2	0	-	2	(100)	0 -
mandibular												
I1	2	0	-	2	(100)	0 -	1	0	-	1	(100)	0 -
I2	6	1	(17)	4	(66)	1 (17)	1	0	-	1	(100)	0 -
C	8	0	-	6	(75)	2 (25)	4	0	-	4	(100)	0 -
PM1	9	2	(22)	7	(78)	0 -	4	0	-	4	(100)	0 -
PM2	7	1	(14)	6	(86)	0 -	4	0	-	4	(100)	0 -
M1	6	0	-	6	(100)	0 -	1	0	-	1	(100)	0 -
M2	7	2	(29)	5	(71)	0 -	4	1	(25)	3	(75)	0 -
M3	5	2	(40)	3	(60)	0 -	6	2	(33)	4	(67)	0 -
total	97	13	(13)	77	(79)	7 (7)	47	3	(6)	41	(88)	3 (6)

N = total number of hypoplastic defects per tooth type
n = number of hypoplastic defects by type
%= frequency of hypoplastic defect types by tooth type

An inter-period comparison of the frequency and inter-tooth distribution of hypoplastic defect types within the permanent dentition.

The greater number of lingual defects in the posterior maxillary teeth of period II may be explained by defects involving both the lingual and labial surfaces. Although an inter-period comparison of defect surface incorporation reveals a similar frequency of defects incorporating the lingual, labial or both the lingual and labial surfaces between the periods, period II does have more maxillary tooth types with hypoplastic defects involving both surfaces (Table 5.8). In contrast, periods I, III and VI have higher inter-tooth frequencies for defects incorporating both surfaces in the mandibular dentition.

Table 5.7: Inter-period comparison of defect presence by surface

	II					I, III & VI				
		lingual		labial			lingual		labial	
	N	n	(%)	n	(%)	N	n	(%)	n	(%)
maxillary										
I1	5	0	-	5	(100)	1	0	-	1	(100)
I2	6	1	(17)	5	(83)	3	0	-	3	(100)
C	7	2	(29)	5	(71)	4	1	(25)	3	(75)
PMI	10	4	(40)	6	(40)	5	0	-	5	(100)
PM2	11	4	(36)	7	(36)	6	2	(33)	4	(67)
M1	8	2	(25)	6	(25)	2	0	-	2	(100)
M2	6	2	(33)	4	(33)	1	0	-	1	(100)
M3	7	2	(29)	5	(29)	2	1	(50)	1	(50)
mandibular										
I1	2	0	-	2	(100)	1	0	-	1	(100)
I2	6	0	-	6	(100)	2	1	(50)	1	(50)
C	11	3	(27)	8	(73)	6	2	(33)	4	(67)
PM1	13	5	(38)	8	(62)	6	2	(33)	4	(67)
PM2	11	5	(45)	6	(55)	7	3	(43)	4	(57)
M1	8	1	(12)	7	(88)	1	0	-	1	(100)
M2	8	2	(25)	6	(75)	5	2	(40)	3	(60)
M3	8	3	(37)	5	(63)	8	2	(25)	6	(75)
total	127	36	(28)	91	(72)	60	16	(27)	44	(73)

N = total number of hypoplastic defects per tooth type present on the lingual or labial surface
n = number of hypoplastic defects by surface
%= frequency of hypoplastic defects by tooth surface

An inter-period comparison of the frequency and distribution of hypoplastic defects present on either the lingual or labial surface. Note defects involving both the lingual and labial surfaces have been counted as one lingual and one labial defect.

A comparison of defect location by tooth third reveals a higher frequency of tooth types exhibiting defects incorporating more than one tooth third in period II. Period II reveals a notable increase in the inter-tooth frequency of hypoplastic defects that involve both the cervical and middle thirds as compared to periods I, III and VI combined (Table 5.9). Period II also provides the only evidence of hypoplastic defects incorporating the entire labial surface.

Table 5.8: Inter-period comparison of hypoplastic defect distribution by surface incorporation

	II						I, III & VI					
		lingual		labial		both		lingual		labial		both
	N	n	(%)	n	(%)	n (%)	N	n	(%)	n	(%)	n (%)
maxillary												
I1	5	0	-	5	(100)	0 -	1	0	-	1	(100)	0 -
I2	5	0	-	4	(80)	1 (20)	3	0	-	3	(100)	0 -
C	6	1	(17)	4	(66)	1 (17)	4	1	(25)	3	(75)	0 -
PMI	7	1	(14)	3	(43)	3 (43)	5	0	-	5	(100)	0 -
PM2	8	1	(12)	4	(50)	3 (38)	4	0	-	2	(50)	2 (50)
M1	6	0	-	4	(67)	2 (33)	2	0	-	2	(100)	0 -
M2	4	0	-	2	(50)	2 (50)	1	0	-	1	(100)	0 -
M3	6	1	(17)	4	(66)	1 (17)	2	1	(50)	1	(50)	0 -
mandibular												
I1	2	0	-	2	(100)	0 -	1	0	-	1	(100)	0 -
I2	6	0	-	6	(100)	0 -	1	0	-	0	-	1 (100)
C	8	0	-	5	(62)	3 (38)	4	0	-	2	(50)	2 (50)
PM1	9	1	(12)	4	(44)	4 (44)	4	0	-	2	(50)	2 (50)
PM2	7	1	(14)	2	(29)	4 (57)	4	0	-	1	(25)	3 (75)
M1	6	0	-	5	(83)	1 (17)	1	0	-	1	(100)	0 -
M2	7	1	(14)	5	(72)	1 (14)	4	1	(25)	2	(50)	1 (25)
M3	5	0	-	2	(40)	3 (60)	6	0	-	4	(67)	2 (33)
total	97	7	(7)	61	(63)	29 (30)	47	3	(6)	31	(66)	13 (28)

N = total number of hypoplastic defects per tooth type
n = number of hypoplastic defects by surface incorporation
%= frequency of hypoplastic defects by surface incorporation

An inter-period comparison of the frequency and distribution of hypoplastic defects involving the lingual, labial/buccal or both surfaces. Note the higher prevalence of defects incorporating both the lingual and labial/buccal surfaces in the maxillary teeth for period II.

Discussion

The results of this study reveal a lower population prevalence of hypoplasia, but a greater frequency of stress episodes, in period II compared to the other periods combined. However, the lack of subadults in periods I, III and VI, and the absence of information on sex and social status

Table 5.9: Defect frequency and distribution in the cervical two-thirds of the crown

	period II				periods I, III & VI			
		cervical	middle	cervical & middle		cervical	middle	cervical & middle
	N	n (%)	n (%)	n (%)	N	n (%)	n (%)	n (%)
maxillary								
I1	5	0 -	1 (20)	4 (80)	1	0 -	1 (100)	0 -
I2	4	0 -	2 (50)	2 (50)	2	1 (50)	1 (50)	0 -
C	4	0 -	0 -	4 (100)	3	1 (33)	1 (33)	1 (33)
PM1	6	2 (33)	4 (67)	0 -	4	1 (25)	3 (75)	0 -
PM2	6	2 (33)	2 (33)	2 (33)	4	2 (50)	2 (50)	0 -
M1	6	3 (50)	3 (50)	0 -	2	1 (50)	1 (50)	0 -
M2	4	3 (75)	0 -	1 (25)	1	0 -	1 (100)	0 -
M3	5	4 (80)	0 -	1 (20)	1	1 (100)	0 -	0 -
mandibular								
I1	2	1 (50)	1 (50)	0 -	1	0 -	1 (100)	0 -
I2	6	1 (17)	3 (50)	2 (33)	1	0 -	1 (100)	0 -
C	7	1 (14)	3 (43)	3 (43)	4	1 (25)	1 (25)	2 (50)
PM1	7	3 (43)	4 (57)	0 -	4	3 (75)	1 (25)	0 -
PM2	6	2 (33)	2 (33)	2 (33)	4	1 (25)	3 (75)	0 -
M1	6	5 (83)	0 -	1 (17)	1	0 -	1 (100)	0 -
M2	6	3 (50)	2 (33)	1 (17)	3	0 -	3 (100)	0 -
M3	3	2 (67)	0 -	1 (33)	4	2 (50)	2 (50)	0 -
total	83	32 (39)	27 (32)	24 (29)	40	14 (35)	23 (58)	3 (7)

N = total number of hypoplastic defects per tooth type
n = number of hypoplastic defects per tooth third location
% = frequency of hypoplastic defects per tooth third location

An inter-period comparison of hypoplastic defect frequency and inter-tooth distribution by cervical or middle tooth third location, or by a combination of cervical and middle thirds. Note the greater frequency of hypoplastic defects incorporating both the cervical and middle tooth thirds in period II.

of the individuals in this study prevent any investigation into which segments of the population are being affected. If the frequency of hypoplasia is considered by the adult group only, then both period II and periods I, III and VI combined exhibit a prevalence of 100%; that is, all adult individuals exhibited one or more hypoplastic teeth. Due to the equally high frequency of stress between periods as indicated by hypoplasia, an inter-tooth comparison of the number of individuals with two or more defects may provide more accurate insight in stress prevalence (e.g. Cucina et al. 1996). However, the present Tell

Leilan dental sample is too small, with individuals represented by incomplete dentitions, for such manipulation.

In the permanent dental sample, the frequency of defective teeth by individual tooth type indicates more hypoplastic maxillary teeth in period II, but the reverse for the mandibular teeth. This is most likely due to the small sample size of periods I, III and VI combined. However, through the calculated defect ratio (i.e. the number of defects per defective tooth), a higher amount of stress is evident for period II. A higher defect ratio translates into a greater mean number of defects per defective tooth. Since hypoplastic defects separated by normal enamel were considered to represent more than one physiological insult, and a defect is regarded as indicative of a stress episode, then an increase in the number of defects should signify a greater number of stress events.

To assess the "impact of stressful events" between populations exhibiting an equally high prevalence of stress, Cucina et al. (1996:6-7) calculated the number of defects relative to the number of defective mandibular canine teeth, and then to the total number of "reliable teeth", and found differing conditions for the five populations under question. Similarly in the Tell Leilan sample, if stress is measured by the number of stress episodes (i.e. defects), then period II exhibits a greater amount of stress, as indicated by number of defects (i.e. defect ratio), in all tooth types except the third molars.

The decrease in stress (i.e. the number of defects) evident in the third molars in period II may provide insight into the pattern of childhood stress. Since the third molars initiate calcification at approximately 7 to 9 years and complete enamel formation at 12 to 16 years (Fuller and Denehy 1984), these findings may represent a decrease in later childhood stress. Recently, through an analysis of inter-tooth patterns of expression of hypoplasia, Wright (1997:233) evaluated the prevailing ecological model for the collapse of the Classic Period Lowland Maya civilization. Although the study did not support the ecological model of collapse, it did note the "...potential of posterior teeth to reveal subtle changes in childhood morbidity when considered individually...".

The higher stress for period II coincides with a higher frequency of tooth types exhibiting hypoplastic defects involving both the lingual and labial surfaces, as well as defects incorporating more than one tooth third. These findings are consistent with those of Smith and Peretz (1986) where an increase in stress for an early Arab population from Dor (Israel) corresponds with an increase in defects occupying more than one tooth third. In light of differential susceptibility to physiological disruption evidenced by intra-tooth analyses (see Goodman and Armelagos 1985b), the increase in the amount of the tooth crown being hypoplastic may indicate an increase in the severity or the duration of stress.

Several studies have attempted to ascertain severity and/or duration of a stress episode (e.g. Blakely and Armelagos 1985; Ensor and Irish 1995; Hutchinson and Larsen 1988) by measuring the width and/or depth of a hypoplastic lesion. Hillson and Bond (1997) have criticized this method on the basis that it does not account for the occlusal to cervical progressive decrease in spacing between developmental layers on the tooth crown. These authors consider counting the developmental layers (i.e. the perikymata) involved in the hypoplastic defect as a more accurate means of assessing the duration of a stress event. With an increase in defects involving both the cervical and middle thirds for most tooth types in period II, this would involve not only an increase in defective crown surface area but more developmental layers as well, and should reflect an increase in the duration, and possibly the severity, of the stress episode.

In sum, more information is needed to ascertain changes in the prevalence of stress for the period preceding abandonment of the site as compared to the other periods. At present, not all archaeological periods are represented by dental remains and those periods that are reveal incomplete dentitions. Ideally, all periods need be represented by a sizable sample to properly investigate changes in the prevalence and distribution of stress. Although hypoplastic data indicates greater stress for period II, this general period preceding the abandonment of Tell Leilan can be divided into subphases; indigenous urbanization on the Habur plains (IIa) and occupation under the Akkadians (IIb). If the specific cultural

influences on health and stress from either of these subphases are ignored, and period II is considered as a whole, then an increase in stress could not be directly related to climate change. A direct comparison of stress frequency and distribution between these two subphases would provide greater insight into the health impact of climate change.

Conclusion

The Tell Leilan dental sample is too small to confirm or refute the claim by Weiss et al. (1993) that climate change is responsible for the abandonment of Tell Leilan. Although these data do suggest a greater frequency of stress episodes (i.e. a higher defect ratio) for the period preceding abandonment of the site (period II), they are insufficient to detect any differences in the population distribution of stress between the periods. In terms of physiological insults, period II reveals not only more stress for early childhood but a possibly less stress in later childhood as compared to the other periods. The greater stress in period II coincides with a shift in the pattern of defect distribution: more tooth types reveal hypoplastic defects incorporating both the labial and lingual surfaces, as well as defects involving more than one tooth third, and may reflect stress episodes of greater duration or severity.

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Chapter 6:
Conclusion

This thesis has investigated the claim of Weiss et al. (1993) - that abrupt climate change is ultimately responsible for the abandonment of the site of Tell Leilan at the end of the third millennium BC - through a multi-level analysis of developmental enamel defects as indicators of non-specific stress. It was hypothesized that the health impact of abrupt climate change at Tell Leilan would be evident in not only an increase in the frequency of stress, but in a greater distribution of stress within the population as well. Although the Tell Leilan dental sample is too small to confirm or refute the conclusion of Weiss et al. (1993), data gained through the analysis of enamel hypoplasia indicate greater stress for the period preceding the abandonment of the site, as compared to previous or subsequent periods. This increase is noted in a higher number of stress episodes experienced by individuals, and may have involved stress of a longer duration or greater severity. Aithough this analysis indicates an increase in the frequency of stress experienced by individuals, it does not demonstrate a greater or lesser distribution within the society. The current Tell Leilan dental sample is too small to assess changes in the distribution of stress between the periods. Data gained from a comparison of the frequency and distribution of enamel hypocalcification conflicts with that from hypoplasia, and may reflect the recording of acquired rather than developmental defects.

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